TRANSACTIONS

OF THE

PATHOLOGICAL SOCIETY OF LONDON.

VOLUME THE TWENTY-SEVENTH.

COMPRISING THE REPORT OF THE PROCEEDINGS FOR THE SESSION 1875-76.

LONDON:
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1876.

The present publication, being the Twenty-seventh Volume of Transactions, constitutes the Thirtieth published Annual Report of the Pathological Society's Proceedings.

The Council think it right to repeat that the exhibitors are alone responsible for the descriptions given of the Specimens exhibited by them, the only change made in the Reports furnished by the authors being such verbal alterations as were absolutely necessary.

53, Berners Street, Oxford Street;
October, 1876.

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OF VOLUME XXVII.

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ELECTED	
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1848	CHARLES ASTON KEY.
1850	PETER MERE LATHAM, M.D.
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1853	BENJAMIN GUY BABINGTON, M.D., F.R.S.
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1873	Sir WILLIAM JENNER, Bart., M.D., K.C.B., D.C.L., F.R.S.
1875	GEORGE D. POLLOCK.

OFFICERS AND COUNCIL

OF THE

Pathological Society of London,

ELECTED AT

THE GENERAL MEETING, JANUARY 4TH, 1876.

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GEORGE D. POLLOCK.

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*** *Members are requested to indicate to the Secretaries corrections when necessary.*

LIST OF MEMBERS OF THE SOCIETY.

Honorary Members.

ARNOTT, JAMES MONCRIEFF, F.R.S., Chapel House, Lady Bank, Fifeshire; and 36, Sussex-gardens, Hyde-park, W.

BERNARD, CLAUDE, M.D., Professor of Physiology in the Faculty of Medicine, Paris.

BILLROTH, THEODOR, M.D., Professor of Surgery in the University of Vienna.

BRUECKE, ERNST, M.D., Professor of Physiology in the University of Vienna.

CRUVEILHIER, J. C., M.D., late Professor in the Faculty of Medicine, Paris.

HELMHOLTZ, H., M.D., Professor of Physiology in the University of Heidelberg.

HENLE, J., M.D., Professor of Anatomy and Physiology in the University of Göttingen.

LUDWIG, C., M.D., Professor of Physiology in the University of Leipzig.

ROKITANKSY, CARL, Baron, M.D., Professor of Pathological Anatomy in the University of Vienna.

STOKES, WILLIAM, M.D., D.C.L., LL.D., F.R.S., M.R.I.A., Regius Professor of Physic in the University of Dublin, Physician in Ordinary to the Queen in Ireland.

VIRCHOW, RUDOLF, M.D., Professor of Pathological Anatomy in the University of Berlin.

VOGEL, JULIUS, M.D., Professor of Pathological Anatomy in the University of Halle.

EXPLANATION OF ABBREVIATIONS.

O.M.—Original Member.	V.-P.—Vice-President.
Pres.—President.	S.—Secretary.
T.—Treasurer.	C.—Member of Council.

Those marked thus (†) have paid Composition Fee for Annual Subscriptions.
Those marked thus (‡) have paid Composition Fee for Transactions.

GENERAL LIST OF MEMBERS.

Elected

1858 ACLAND, HENRY WENTWORTH, M.D., F.R.S., Regius Professor of Medicine, University of Oxford, Physician to the Radcliffe Infirmary, Oxford.

‡1866 ADAMS, ARTHUR BAYLEY.

1869 ADAMS, JAMES EDWARD, Surgeon to the London Hospital, 10, Finsbury-circus, E.C.

O.M. ADAMS, WILLIAM, Consulting Surgeon to the National Orthopædic Hospital, 5, Henrietta-street, Cavendish-square, W. (C. 1851-4. V.-P. 1867-9.)

Elected

1859 Adams, William, 37, Harrington-square, N.W.

1848 Aikin, Charles A., 7, Clifton-place, Sussex-square, Hyde-park, W. (C. 1861-6.)

1872 Aikin, Charles Edmund, 7, Clifton-place, Sussex-square, Hyde-park, W.

1871 Air, A. Cummings, 88, Kennington-park-road, S.E.

1869 Allbutt, Thomas Clifford, M.D., Physician to the Leeds General Infirmary, 35, Park-square, Leeds.

1868 Anderson, J. Ford, M.D., 28, Buckland-crescent, Belsize-park, N.W.

1871 Anderson, William, Professor of Medical Science at the University of Yeddo, Japan.

1859 Andrew, Edwyn, M.D., Hardwick House, St. John's-hill, Shrewsbury.

1863 Andrew, James, M.D., Physician to St. Bartholomew's Hospital, 22, Harley-street, W. (C. 1868-70.)

1866 Arnott, Henry. (C. 1872, 1875-6. S. 1873, 1874.)

1863 Bagshawe, Frederick, M.A., M.D., 16, Warrior-square, Hastings.

1864 Baker, William Morrant (C.), Assistant Surgeon to, and Lecturer on Physiology at, St. Bartholomew's Hospital, 26, Wimpole-street, Cavendish-square, W. (C. 1873-6.)

‡1856 Balding, Daniel Barley, Royston, Herts.

1851 Barclay, A. Whyte, M.D., Physician to St. George's Hospital, 23A, Bruton-street, Berkeley-square, W. (C. 1858-61.)

1875 Barker, Arthur, 16, Kensington-park-road, W.

1874 Barlow, Thomas, M.D., B.S., Assistant Physician to Charing Cross Hospital and to the Children's Hospital, 10, Montague-street, Russell Square.

1871 Barnes, Robert, M.D., Obstetric Physician to St. George's Hospital, 31, Grosvenor-street, W.

1876 Barnes, R. S. Fancourt, M.B., C.M., 39, Weymouth-street, Portland-place, W.

1862 Barratt, Joseph Gillman, M.D., Accoucheur to the St. George's and St. James's Dispensary, 8, Cleveland-gardens, Bayswater, W.

1853 Barwell, Richard, Surgeon to the Charing Cross Hospital, 32, George-street, Hanover-square, W. (C. 1862-4.)

1857 Basham, William R., M.D., Senior Physician to the Westminster Hospital, 17, Chester-street, Belgrave-square, S.W.

1861 Bastian, H. Charlton, M.A., M.D., F.R.S., Professor of Pathological Anatomy in University College, and Physician to University College Hospital, 20, Queen Anne-street, W. (C. 1869-71.)

†1876 Batteson, John, Medical Officer of the Royal Humane Society; 1, Coborn-place, Bow-road, E.

1870 Bäumler, Christian G. H., M.D., Professor of Materia Medica in the University of Erlangen.

1871 Baxter, Evan Buchanan, M.D., Professor of Materia Medica, King's College, London, and Assistant Physician to King's College Hospital, 28, Weymouth-street, Portland-place, W.

Elected.

1874 Beach, Fletcher, M.B., Asylum for Idiots, Lower Clapton, N.E.

1852 Beale, Lionel S., M.B., F.R.S., Physician to King's College Hospital, 61, Grosvenor-street, W. (C. 1858-9. V.-P. 1874-5.)

1856 Bealey, Adam, M.D., M.A., Oak-lea, Harrogate.

1870 Beck, Marcus, M.S. (C.), Assistant Surgeon to University College Hospital, 30, Wimpole-street, Cavendish-square, W. (C. 1875-6.)

1853 Beck, Thomas Snow, M.D., F.R.S., 7, Portland-place, W.

1865 Beeby, Walter, M.D., Bromley, Kent.

1865 Beigel, Hermann, M.D., 2, Lichtensteinstrasse, Vienna.

1875 Bell, H. Royes, 44, Harley-street, Cavendish-square, W.

1865 Bellamy, Edward (C.), Senior Assistant Surgeon to the Charing Cross Hospital, 59, Margaret-street, Cavendish-square, W. (C. 1876.)

1847 Bennet, James Henry, M.D., Weybridge, Surrey.

1876 Bennett, Alex. Hughes, M.D., Medical Registrar to the Westminster Hospital, 18, St. George's-terrace, Gloster-road.

O.M. Bennett, James Risdon, M.D., F.R.S., Consulting Physician to St. Thomas's Hospital, and to the City of London Hospital for Diseases of the Chest, 22, Cavendish-square, W. (C. 1846-8. V.-P. 1856-9.)

1876 Bernays, Sidney, Acre House, Brixton.

‡1856 Bickersteth, Edward R., Surgeon to the Liverpool Royal Infirmary, 2, Rodney-street, Liverpool.

1850 Birkett, Edmund Lloyd, M.D., Consulting Physician to the City of London Hospital for Diseases of the Chest, 48, Russell-square, W.C. (C. 1856-7.)

O.M. Birkett, John, Consulting Surgeon to Guy's Hospital, 59, Green-street, Grosvenor-square, W. (C. 1851. V.-P. 1860-2.)

1865 Bisshopp, James, Bedford-place, Tunbridge Wells.

1853 Black, Cornelius, M.D., Physician to the Chesterfield Dispensary, St. Mary's-gate, Chesterfield.

1850 Blagden, Robert, Stroud, Gloucestershire.

1863 Blanchet, Jean B., M.D., M.S., Montreal, Quebec, Canada.

1876 Blasson, William, Edgeware, Middlesex.

1872 Bloxam, John Astley, Assistant Surgeon to Charing Cross Hospital, 8, George-street, Hanover-square, W.

1876 Bond, Thomas, M.B., 50, Parliament-street, S.W.

1869 Bourne, Walter, M.D.

1861 Bower, Richard Norris, 14, Doughty-street, Mecklenburg-square, W.C.

1851 Bowman, William, F.R.S., Surgeon to the Royal Ophthalmic Hospital, 5, Clifford-street, Bond-street, W. (C. 1855-6.)

‡1867 Bridgewater, Thomas, M.B. Lond., Harrow-on-the-hill, Middlesex.

1873 Briggs, Jacob Myers, M.D., Coeymans, New York, U.S.

1868 Bright, G. C., M.B., 29, Lüttichanstrasse, Dresden.

1857 Briscoe, John, 12, Broad-street, Oxford.

†1851 Bristowe, John S., M.D., Physician to, and Lecturer on the Theory and Practice of Medicine at, St. Thomas's Hospital, 11, Old Burlington-street, W. (C. 1854-8. S. 1861-4. C. 1865-7. V.-P. 1868-70.)

Elected

1860 Broadbent, William Henry, M.D. Lond., Physician to St. Mary's Hospital, and Physician to the London Fever Hospital, 34, Seymour-street, Portman-square, W. (C. 1871-3.)

1852 Brodhurst, Bernard E., Surgeon to the Royal Orthopædic Hospital, 20, Grosvenor-street, W. (C. 1862-4.)

1863 Brodie, George Bernard, M.D., Consulting Physician-Accoucheur to Queen Charlotte's Hospital, 56, Curzon-street, Mayfair, W.

1846 Brooke, Charles, M.B., F.R.S., Consulting Surgeon to the Westminster Hospital, 16, Fitzroy-square, W. (C. 1853-5. V.-P. 1864-5.)

1865 Brown, Augustus, M.D., 29, Belitha-villas, Barnsbury-park, N.

1871 Brown, Frederick Gordon, 15, Finsbury-circus, E.C.

1875 Browne, George Buckstone, 15, Bulstrode-street.

1866 Browne, Lennox, Surgeon to the Central Throat and Ear Hospital, and to the Royal Society of Musicians, 36, Weymouth-street, Portland-Place, W.

O.M. Browne, Joseph Hullett, M.D., Physician to the St. Pancras Royal General Dispensary, 55, Gordon-square, W.C. (C. 1859-60.)

1855 Bryant, Thomas, Surgeon to Guy's Hospital, 53, Upper Brook-street, Grosvenor-square, W. (C. 1863-6.)

1854 Buchanan, George, M.D., Medical Inspector to the Privy Council, 24, Nottingham-place, Marylebone-road, W. (C. 1864-6.)

1862 Buchanan, Albert, M.B. Lond., 382, Camden-road, N.

1858 Budd, George, M.D., F.R.S., Ashleigh, Barnstaple. (C. 1862-4.)

1860 Burton, Alfred, 13, Dover-street, Piccadilly, W.

1853 Burton, John M., Lee-park Lodge, Lee, Kent, S.E.

O.M. Busk, George, F.R.S., Consulting Surgeon to the Seamen's Hospital, Greenwich, 32, Harley-street, Cavendish-square, W.C. C. 1846-8. V.-P. 1858-60.)

1872 Butlin, Henry Trentham (C.), Surgical Registrar to St. Bartholomew's Hospital, Assistant Surgeon to the West London Hospital, 47, Queen Anne-street, W. (C. 1876.)

1866 Butt, William Frederick, 12, South-street, Park-lane, W.

1856 Buzzard, Thomas, M.D., Physician to the National Hospital for the Epileptic and Paralysed, 56, Grosvenor-street, W. (C. 1869-70.)

1856 Callender, G. W., F.R.S., Surgeon to St. Bartholomew's Hospital, 7, Queen Anne-street, Cavendish-square, W. (C. 1865-9.)

†O.M. Camps, William, M.D. (C. 1856-9.)

‡1855 Carpenter, Alfred, M.D., High-street, Croydon.

1872 Carr, William, M.D., Lee-grove, Blackheath, S.E.

1871 Carter, Charles Henry, M.D., B.S. Lond., Physician to the Hospital for Women, 45, Great Cumberland-place, Hyde-park, W.

Elected

1855 Carter, H. Vandyke, M.D., Professor of Anatomy and Physiology, Grant Medical College, Bombay. [22, Clarendon-road, Victoria-road, Kensington, W.]

1876 Carter, Robert Brudenell, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. George's Hospital, 69, Wimpole-street, Cavendish-square, W.

†1868 Cavafy, John, M.D., Assistant Physician to, and Lecturer on Physiology at, St. George's Hospital, Physician to the Victoria Hospital for Children, 2, Upper Berkeley-street, Portman-square, W.

1864 Cay, Charles Vidler, Coldstream Guards, the Hospital, Vincent-square, Westminster, S.W.

1863 Cayley, William, M.D. (C.), Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital, 58, Welbeck-street, Cavendish-square, W. (C. 1870-1, 1875-6. S. 1872-4.)

1869 Chaffers, Edward, Keighley, Yorkshire.

1849 Chalk, William Oliver, 3, Nottingham-terrace, Regent's-park, N.W. (C. 1856-7.)

1876 Charles, T. W. Cranstoun, M.D., M.C., Medical Registrar, St. Thomas's Hospital.

1870 Cheadle, Walter Butler, M.D., Assistant Physician to St. Mary's Hospital, 2, Hyde-park-place, Cumberland-gate, W.

O.M. Chevers, Norman, M.D., India. (C. 1848.)

1872 Cheyne, William Romley, 27, Nottingham-place, Marylebone-road, W.

‡1858 Child, Gilbert W., 11, Norham-gardens, Oxford.

1873 Chisholm, Edwin, Camden-by-Sydney, New South Wales.

1855 Cholmeley, William, M.D., Physician to the Great Northern Hospital, and to the Margaret-street Infirmary for Consumption, 63, Grosvenor-street, W. (C. 1871-3.)

1871 Christie, Thomas Beath, M.D., Superintendent of the Royal India Asylum, Ealing, Middlesex.

1865 Church, William Selby, M.D., Physician to St. Bartholomew's Hospital, 130, Harley-street, Cavendish-square, W. (C. 1871-3.)

†1868 Churchill, Frederick, M.B., Assistant Surgeon to the Victoria Hospital for Children, 6, Sumner Place, S.W.

1861 Clapton, Edward, M.D., 10A, St. Thomas's-street, Southwark, S.E.

1854 Clark, Andrew, M.D., Physician to the London Hospital, 16, Cavendish-square, W. (C. 1862-5.)

1872 Clark, Andrew, Assistant Surgeon to the Middlesex Hospital, 19, Cavendish Place, W.

1865 Clarke, Jacob Lockhart, M.D., F.R.S., 64, Harley-street, Cavendish-square, W. (C. 1868-70.)

1867 Clarke, William Fairlie, M.A., Southborough, Tunbridge Wells. (C. 1873-5.)

‡1875 Clarkson, John, Surgeon in the India Department, Bombay Presidency, India.

Elected

1875 Clutton, Henry Hugh, Resident Assistant Surgeon, St. Thomas's Hospital.

‡1865 Coates, Charles, M.D., Physician to the Bath General and Royal United Hospitals, 10, Circus, Bath.

O.M. Cock, Edward, Consulting Surgeon to Guy's Hospital, 36, Dean-street, south, Tooley-street, S.E. (C. 1846-8. V.-P. 1856.)

1856 Cockle, John, M.D., M.A., Physician to the Royal Free Hospital, 7, Suffolk-place, Pall-mall, S.W.

O.M. Cohen, Daniel Whitaker, M.D., South-bank, Northdown-lane, Bideford, Devon.

†1866 Coles, George Charles, Surgeon to the Infirmary for Epilepsy and Paralysis, and Assistant Surgeon to the Royal South London and Central London Ophthalmic Hospitals, 20, Great Coram-street, Russell-square, W.C.

Colley, see Davies-Colley.

1858 Cooke, Robert Thomas, Surgeon to the Scarborough Dispensary, 15, St. Nicholas-cliff, Scarborough, Yorkshire.

1871 Cooke, Thomas, Assistant Surgeon to the Westminster Hospital, 16, Woburn-place, W.C.

1866 Coombs, Rowland Hill, Mill-street, Bedford.

1851 Cooper, William White, Consulting Ophthalmic Surgeon to St. Mary's Hospital, 19, Berkeley-square, W. (C. 1860-2.)

1853 Cornish, William Robert, Surgeon-Major, Madras Army, Sanitary Commissioner for Madras.

1875 Cory, Robert, M.B., Assistant Obstetric Physician to St. Thomas's Hospital, 14, Palace-road, Albert Embankment, S.E.

1859 Coulson, Walter J., Surgeon to the Lock Hospital, 17, Harley-street, Cavendish-square, W.

O.M. Coulson, William, Consulting Surgeon to St. Mary's Hospital, 1, Chester-terrace, Regent's-park, N.W. (C. 1850-3. V.-P. 1862-3.)

†1861 Couper, John, Surgeon to the London Hospital, 80, Grosvenor-street, Grosvenor-square, W. (C. 1870-2.)

1873 Coupland, Sidney, M.D., Assistant Physician to the Middlesex Hospital, 6, Old Cavendish-street, Cavendish-square, W.

1873 Cripps, William Harrison, 53A, Pall-mall.

O.M. Crisp, Edwards, M.D., 29, Beaufort-street, Chelsea, S.W. (C. 1846-7. V.-P. 1870-2.)

1848 Critchett, George, Surgeon to the Royal London Ophthalmic Hospital, Moorfields, and Ophthalmic Surgeon to the Middlesex Hospital, 21, Harley-street, W. (S. 1849. C. 1851, 1858-9. V.-P. 1866-7.)

1856 Croft, John, Surgeon to St. Thomas's Hospital, 61, Brook-street, Grosvenor-square, W. (C. 1870-2.)

‡1866 Cromarty, James Pattison, Civil Surgeon, Tavoy, Burmah. [Agents: Messrs. Fergusson & Co., 77, Clive-street, Calcutta.]

1861 Crosby, Thomas Boor, M.D., 21, Gordon-square, W.C.

Elected

1875 Cross, Francis Richardson, King's College Hospital.

1854 Cross, Robert, M.D., 42, Craven-street, Strand, W.C.

1864 Crucknell, Henry H., M.B. (C.), Physician to the Great Northern Hospital, and to the Royal Infirmary for Diseases of the Chest, City-road [care of Geo. Pitman, Esq., 9, Worcester-road, Sutton, Surrey].

1871 Cumberbatch, Elkin, Demonstrator of Anatomy at St. Bartholomew's Hospital, 17, Queen Anne-street, W.

1858 Cumberbatch, Laurence T., M.D., 25, Cadogan-place, Sloane-street, S.W.

1855 Curling, Thomas Blizard, F.R.S., Consulting Surgeon to the London Hospital, 39, Grosvenor-street, W. (C. 1857-60. V.-P. 1866-8.)

1873 Curnow, John, M.D., Professor of Anatomy at King's College, and Assistant Physician to King's College Hospital, 3, Warwick-street, Cockspur-street, S.W.

‡1865 Curran, William, M.D., Army Medical Staff. [Agent: Mr. H. K. Lewis, 136, Gower-street, W.C.]

1863 Dane, Thomas, 86, Finchley-road, N.W.

1873 Davidson, Alexander, M.D., Physician to the Liverpool Northern Hospital, 49, Rodney-street, Liverpool.

1869 Davies-Colley, J. N., M.B., Assistant Surgeon to Guy's Hospital, 36, Harley-street, Cavendish-square.

O.M. Davies, Herbert, M.D., Consulting Physician to the Infirmary for Asthma, &c., and Consulting Physician to the London Hospital, 23, Finsbury-square, E.C. (C. 1849-50. V.-P. 1871.)

1847 Davis, John Hall, M.D., Physician-Accoucheur to the Middlesex Hospital, and to the Royal Maternity Charity, 24, Harley-street, Cavendish-square, W. (C. 1852-3.)

‡1859 Davis, Francis William, R.N., Surgeon to the Naval Medical Establishment, Lisbon; 11 and 12, Love-lane, Aldermanbury, E.C.

1867 Davy, Richard, Surgeon to the Westminster Hospital, 33, Welbeck-street Cavendish-square, W.

1866 Day, William Henry, M.D., Physician to the Samaritan Free Hospital for Women and Children, 10, Manchester-square, W.

1872 Decastro, James C., M.B., 38, Rutland-gate, Knightsbridge, S.W., and Pau, France.

1871 De Liefde Temple, John, M.D. [per Mr. James Nimmo, 7, Red Lion-court, Watling-street, E.C.].

1863 Devereux, Daniel, Tewkesbury.

1856 Dick, H., M.D., 59, Wimpole-street, Cavendish-square, W.

1871 Dickinson, Edward Harriman, M.B., Physician to the Liverpool Northern Hospital, 162, Bedford-street, Liverpool.

1858 Dickinson, William Howship, M.D., Physician to the Hospital for Sick Children, Physician and Lecturer on Pathology to St. George's Hospital, 11, Chesterfield-street, Mayfair, W. (C. 1866-8. S. 1869-71. V.-P. 1872-4.)

Elected

1872 Diver, Ebenezer, M.D., Kenley, Caterham-valley, Surrey.

O.M. Dixon, James, Consulting Surgeon to the Royal Ophthalmic Hospital, Moorfields, Harrowlands, Dorking, Surrey. (C. 1852-6. V.-P. 1860-2.)

1874 Donkin, H. B., M.B., Assistant-Physician to the Westminster Hospital, 50, Harley-street, Cavendish-square, W.

1872 Doran, Alban Henry Griffiths, Surgeon to the Metropolitan and the Westminster Dispensaries, 33, Lansdowne-road, Notting-hill, W.

†1866 Down, John Langdon H., M.D., Physician to the London Hospital, 39, Welbeck-street, Cavendish-square, W. (C. 1872-4.)

1872 Dowse, Thomas Stretch, M.D., Central London Sick Asylum, Highgate, N., and 9, Spring-gardens, S.W.

1866 Drewry, George Overend, M.D., 15, Queen Anne-street, W.

1865 Duckworth, Dyce, M.D., Assistant Physician to St. Bartholomew's Hospital, 11, Grafton-street, Bond-street, W.

1863 Dudfield, Thomas Orme, M.D., 8, Upper Phillimore-place, Kensington, W.

1847 Dudgeon, Robert E., M.D., 53, Montagu-square, W.

1852 Duff, George, M.D., High-street, Elgin.

1865 Duffin, Alfred Baynard, M.D., Physician to King's College Hospital, 18, Devonshire-street, Portland-place, W. (C. 1872-4.)

1875 Duka, Theodore, M.D., Surgeon-Major, H.M.'s Bengal Army; 38, Montagu-square, W.

1868 Duke, Oliver Thomas, M.B., India.

1871 Dukes, Clement, M.B., B.S., Horton-crescent, Rugby.

1861 Dunn, Robert William, 13, Surrey-street, Strand, W.C.

1865 Du Pasquier, Claudius Francis, Surgeon-Apothecary to the Queen, 62, Pall-mall, S.W.

1858 Durham, Arthur Edward, Surgeon to Guy's Hospital, 82, Brook-street, Grosvenor-square, W. (C. 1869-71.)

1867 Edis, Arthur W., M.D., Assistant Obstetric Physician to the Middlesex Hospital, 22, Wimpole-street, Cavendish-square, W.

1867 Ellis, James, M.D., Belle-grove Villa, Welling, Blackheath, S.E.

1847 Ellis, James, Sudbrook-park, Richmond, Surrey. Agent: (Tweedie, 337, Strand.)

1873 Engelmann, George Julius, M.D., A.M., 3003, Locust-street, St. Louis, Miss., U.S.

1846 Erichsen, John Eric, F.R.S., Surgeon to University College Hospital, 6, Cavendish-place, Cavendish-square, W. (C. 1849-51. V.-P. 1863-4.)

1853 Evans, Conway, M.D., 5, Tavistock-street, Covent-garden, W. C. (C. 1867-8.)

1873 Evans, George Henry, M.D., Assistant Physician to the Middlesex Hospital, 29, Devonshire-street, Portland-place.

Elected

1875 Evans, Julian, A.M., M.D., Physician to the Victoria Hospital for Sick Children, 123, Finboro'-road, Redcliffe-square, West Brompton, S.W.

1876 Ewart, James Cosar, M.B., C.M., University College Hospital.

‡1859 Ewens, John, Barton Lodge, Cerne Abbas, Dorset.

1864 Fagge, Charles Hilton, M.D., Assistant Physician to, and Lecturer on Pathology at, Guy's Hospital, 11, St. Thomas's-street, Southwark, S.E. (C. 1870-2.)

1862 Farquharson, Robert, M.D. (C.), Lecturer on Materia Medica at St. Mary's Hospital, 23, Brook-street, Grosvenor-square, W. (C. 1876.)

1872 Fayrer, Sir Joseph, C.S.I., M.D., F.R.S. Ed., Hon. Physician to the Queen, Surgeon-Major, Bengal Army, Examining Medical Officer to the Secretary of State for India in Council, 16, Granville-place, Portman-square, W.

1872 Fenn, Edward L., M.B., The Old Palace, Richmond.

1872 Fenwick, John C. J., M.D. [41, Oxford-terrace, Hyde-park, W.].

1863 Fenwick, Samuel, M.D., Physician, with charge of out-patients, to the London Hospital, 29, Harley-street, W.

1848 Fergusson, Sir William, Bart., F.R.S., Surgeon to King's College Hospital, 16, George-street, Hanover-square, W. (C. 1849–50. V.-P. 1851–8. *Pres.* 1859–60. V.-P. 1861.)

1846 Fincham, George T., M.D., Physician to the Westminster Hospital, 13, Belgrave-road, S.W. (C. 1855.)

1870 Fish, John Crockett, M.B., 92, Wimpole-street, W.

1859 Fisher, Alexander, M.D., Assistant Surgeon, R.N., Her Majesty's Ship "Endymion."

1874 Fisher, Fred. R., Victoria Hospital for Sick Children, 79, Grosvenor-street, W.

1855 Flower, William H., F.R.S., Conservator of the Museum, Royal College of Surgeons, 39, Lincoln's-inn-fields, W.C. (C. 1862–4.)

1872 Forbes, Daniel Mackay, L.R.C.P. Ed., Shoreditch Workhouse, Kingsland.

1852 Forbes, J. Gregory, 82, Oxford-terrace, Hyde-park, W. (C. 1860-3.)

†O.M. Forster, John Cooper, Surgeon to Guy's Hospital, 29, Upper Grosvenor-street, W. (C. 1857-8. V.-P. 1871-3.)

‡1866 Foster, Balthazar Walter, M.D., Physician to the General Hospital, Birmingham, 16, Temple-row, Birmingham.

1872 Fotherby, Henry J., M.D., Physician to the Metropolitan Free Hospital, 3, Finsbury-square, E.C.

1862 Fox, Wilson, M.D. (V.P.), Holme Professor of Clinical Medicine in University College, and Physician to University College Hospital, 67, Grosvenor-street, W. (C. 1868-70. V.-P. 1875-6.)

1865 Fox, W. Tilbury, M.D., Physician to the Skin Department of University College Hospital, 14, Harley-street, W.

Elected

1858 Francis, Charles Richard, M.B., Bengal Medical Establishment, Indian Army.

1875 Franklin, George Cooper, Leicester.

O.M. Frere, J. C.

1864 Frodsham, John Mill, M.D., Streatham, S.W.

‡1858 Gairdner, William Tennant, M.D., Professor of Medicine in the University of Glasgow, 225, St. Vincent-street, Glasgow.

1870 Galton, Edmund H., Springfield House, Brixton-hill, S.W.

1870 Galton, John H., M.D., 1, Woodside, Anerley-road, Upper Norwood, S.E.

1855 Gamgee, Joseph Sampson, Surgeon to the Queen's Hospital, Birmingham, 20, Broad-street, Birmingham.

1855 Gamgee, J.

1846 Garrod, Alfred Baring, M.D., F.R.S., Consulting Physician to King's College Hospital, 10, Harley-street, Cavendish-square, W. (C. 1851. V.-P. 1863-5.)

1872 Garton, William, Hardshaw-street, St. Helen's, Lancashire.

O.M. Gay, John, Senior Surgeon to the Great Northern Hospital, 10, Finsbury-place South, E.C. (C. 1852-4. V.-P. 1870-2.)

1853 Gibbon, Septimus, M.D., 39, Oxford-terrace, Hyde-park, W.

1876 Gill, John, M.D., 19, Sackville-street, Piccadilly; and 17, Cedars-road, Clapham-common, S.W.

1873 Godlee, Rickman John, M.B., B.S., Assistant Surgeon to University College Hospital, 22, Henrietta-street, Cavendish-square, W.

1875 Godson, Clement, M.D., 8, Upper Brook-street, Grosvenor-square.

1871 Goodhart, James Frederick, M.D. (C.), Medical Registrar to Guy's Hospital, and Demonstrator of Morbid Anatomy, 27, Weymouth-street, Portland-place, W. (C. 1876.)

1875 Gould, Alfred Pearce, M.B., Surgical Registrar to University College Hospital, 93, Gower-street, W.C.

1870 Gowers, William Richard, M.D., Assistant Physician to University College Hospital, 50, Queen Anne-street, W.

1858 Gowland, Peter Y., Surgeon to St. Mark's Hospital, 34, Finsbury-square, E.C.

1867 Green, T. Henry, M.D. (Hon. Secretary), Physician to Charing Cross Hospital, Assistant Physician to the Hospital for Consumption, Brompton, 74, Wimpole-street, W. (C. 1871-3. S. 1875-6.)

1873 Greenfield, William Smith, M.B., B.S., Lecturer on Morbid Anatomy, St. Thomas's Hospital, Physician to the Royal Hospital for Diseases of the Chest, City-road, 93, Wimpole-street, W.

1856 Greenhalgh, Robert, M.D., Physician-Accoucheur to St. Bartholomew's Hospital, 72, Grosvenor-street, W.

‡1855 Greenhill, William Alexander, M.D., Carlisle-parade, Hastings.

Elected

1863 Greenhow, Edward Headlam, M.D., F.R.S., Physician to the Middlesex Hospital, 14a, Manchester-square, W. (C. 1867-9.)

1871 Grigg, William Chapman, M.D., Assistant Obstetric Physician to the Westminster Hospital, and Physician to the In-Patients, Queen Charlotte's Lying-in Hospital, 6, Curzon-street, Mayfair, W.

1861 Gueneau de Mussy, Henri, M.D., 15, Rue du Cirque, Paris.

1863 Gull, Sir William Withey, Bart., M.D., D.C.L., F.R.S., Consulting Physician to Guy's Hospital, 74, Brook-street, Grosvenor-square, W.

1876 Gwyther, James, M.B. Lond., St. Mary Church, Torquay.

1849-59 Habershon, Samuel Osborne, M.D., Physician to, and Lecturer on Medicine at, Guy's Hospital, 70, Brook-street, Grosvenor-square, W. (Re-elected 1874.) (C. 1855-6.)

1851 Hacon, E. Dennis, 249, Mare-street, Hackney, N.E. (C. 1872.)

1848 Hare, Charles John, M.D. (V.-P.), late Physician to University College Hospital, 57, Brook-street, Grosvenor-square, W. (C. 1852-4. V.-P. 1874-6.)

†1856 Harley, George, M.D., F.R.S., 25, Harley-street, Cavendish-square, W. (C. 1862-5.)

1863 Harling, Robert Dawson, M.D. Lond., 16, Seymour-street, Portman-square, W.

1872 Harris, Henry, M.D., Trengweath-place, Redruth, Cornwall.

†1858 Hart, Ernest, 37, Great Queen-street, Lincoln's-inn-fields, W.C. (C. 1867-8.)

†1859 Hastings, Cecil William, M.B., 13, Queen Anne-street, Cavendish-square, W.

1870 Haward, John Warrington, Assistant Surgeon to St. George's Hospital, 5, Montagu-street, Portman-square, W.

O.M. Hawkins, Cæsar H., F.R.S., Consulting Surgeon to St. George's Hospital, 26, Grosvenor-street, W. (V.-P. 1846-51. *Pres.* 1852-3.)

1857 Hawksley, Thomas, M.D., Physician to the Margaret-street Dispensary for Consumption, 6, Brook-street, Hanover-square, W.

1869 Hay, Thomas Bell, L.R.C.P. Ed., Christchurch, New Zealand.

1856 Heath, Christopher, Holme Professor of Clinical Surgery in University College, and Surgeon to University College Hospital, 36, Cavendish-square, W. (C. 1866-7.)

1869 Hensley, Philip J., M.D., Assistant Physician to St. Bartholomew's Hospital, 4, Henrietta-street, Cavendish-square, W.

‡1868 Heslop, Thomas P., M.D., Physician to the Children's Hospital, Birmingham.

O.M. Hewett, Prescott G., F.R.S., Consulting Surgeon to St. George's Hospital, 1, Chesterfield-street, Mayfair, W. (C. 1846-52. V.-P 1854-7. *Pres.* 1863-4. V.-P. 1865-8.)

1855 Hewitt, Graily, M.D., Obstetric Physician to University College Hospital, 36, Berkeley-square, W. (C. 1865-7.)

Elected

1864 Hickman, William, M.B., Surgeon to the Samaritan Free Hospital, 1, Dorset-square, N.W.

1860 Hill, M. Berkeley, M.B., Surgeon to University College Hospital, and Surgeon for Out-Patients to the Lock Hospital, 55, Wimpole-street, Cavendish-square, W. (C. 1874-5.)

1867 Hill, Samuel, M.D., 22, Mecklenburg-square, W.C.

†O.M. Hilton, John, F.R.S., Consulting Surgeon to Guy's Hospital, 10, New Broad-street, E.C. (C. 1848-50. V.-P. 1863-4, 1873-4. *Pres.* 1871-2.)

1875 Hitchcock, Harry Knight, St. Clare College Park, Lee, Lewisham, S.E.

1852 Hogg, Jabez, Surgeon to the Westminster Ophthalmic Hospital, 1, Bedford-square, W.C. (C. 1860-2.)

1874 Hoggan, George, M.B., 13, Granville-place, Portman-square, W.

1847 Holman, H. Martin, M.D., Hurstpierpoint, Sussex.

1854 Holmes, Timothy, Surgeon-in-Chief to the Metropolitan Police, Surgeon to St. George's Hospital, 18, Great Cumberland-place, Hyde-park, W. (C. 1862-3. S. 1864-7. C. 1868. V.-P. 1869-71.)

1850 Holt, Barnard Wight, Consulting Surgeon to the Westminster Hospital, 14, Savile-row, W. (C. 1853.)

O.M. Holthouse, Carsten, 7, George-street, Hanover-square, W. (C. 1852-4. V.-P. 1874-5.)

1864 Hood, Wharton P., M.D., 65, Upper Berkeley-street, Portman-square, W.

1865 Hooper, John Harwood, M.B., Auckland, New Zealand.

1870 Hope, William, M.D., 5, Bolton-row, Mayfair, W.

1866 Howard, Edward, M.D.

1875 Howse, Henry Greenway, M.S., Surgeon to Guy's Hospital, and to the Evelina Hospital for Sick Children, 10, St. Thomas's-street, S.E.

†1856 Hudson, John, M.D., 11, Cork-street, Bond-street, W.

1854 Hulke, John Whitaker, F.R.S. (V.-P.), Surgeon to the Middlesex Hospital, and Surgeon to the Royal London Ophthalmic Hospital, 10, Old Burlington-street, W. (C. 1863-5. S. 1868-72. V.-P. 1873-6.)

1854 Hulme, Edward Charles, Woodbridge-road, Guildford.

1853 Humby, Edwin, M.D., 83, Hamilton-terrace, St. John's Wood, N.W.

1874 Humphreys, Henry, M.D., Medical Registrar, Middlesex Hospital.

1866 Hunter, Charles, 30, Wilton-place, Belgrave-square, S.W.

1852 Hutchinson, Jonathan, Surgeon to the London Hospital, and to the Royal London Ophthalmic Hospital, Moorfields, 15, Cavendish-square, W. (C. 1856-9. V.-P. 1872-3.)

1875 Irvine, J. Pearson, M.D., 3, Mansfield-street, Cavendish-square.

1865 Jackson, J. Hughlings, M.D., Physician to the London Hospital, Physician to the National Hospital for the Paralysed and Epileptic, 3, Manchester-square, W. (C. 1872-3.)

Elected

1859 Jackson, Thomas Carr (C.), Surgeon to the Great Northern Hospital, 91, Harley-street, Cavendish-square, W. (C. 1875-6.)

1876 Jackson, Ernest Carr, St. Mark's Hospital, City-road.

1875 Jalland, William Hamerton, 34, Botham, York.

‡1853 Jardine, John Lee, Capel, near Dorking, Surrey.

1847 Jay, Edward, 112, Park-street, Grosvenor-square, W.

O.M. Jenner, Sir William, Bart., M.D., D.C.L., K.C.B., F.R.S. (V.-P., late President), Physician to University College Hospital, 63, Brook-street, Grosvenor-square, W. (C. 1850-3. V.-P. 1862-4, 1875-6. *Pres.* 1873-4.)

1875 Jessett, Frederic Bowreman, Pier-road, Erith, Kent.

1866 Jessop, Thomas Richard, 31, Park-square, Leeds.

1876 Johnson, Charles Henry, late Staff Surgeon, Turkish Contingent, 18, Westbourne-place, Eaton-square, S.W.

O.M. Johnson, George, M.D., F.R.S., Physician to King's College Hospital, 11, Savile-row, W. (C. 1846-50. V.-P. 1863-4.)

1854 Johnstone, Athol A. W., St. Moritz House, 61, Dyke-road, Brighton.

1853 Jones, Sydney, M.B., Surgeon to St. Thomas's Hospital, 10*b*, St. Thomas's-street, Southwark, S.E. (C. 1864-6.)

1862 Jones, Thomas Ridge, M.D., Physician to the Victoria Hospital for Sick Children, 19, Chapel-street, Belgrave-square, S.W.

1858 Jones, William Price, M.D., Claremont-road, Surbiton, Kingston.

1860 Jones, Walter, College-yard, Worcester.

1867 Kelly, Charles, M.D., Medical Officer of Health for the West Sussex Combined Sanitary District, Worthing, Sussex. (C. 1874.)

1846 Kent, Thomas J., 60, St. James's-street, S.W.

1852 Kershaw, W. Wayland, M.D., Kingston-on-Thames.

1872 Kesteven, William B., M.D., 401, Holloway-road, N.

1859 Kiallmark, Henry Walter (C.), 66, Prince's-square, Bayswater, W. (C. 1875-6.)

1867 King, Edwin Holborow, 18, Stratford-place, Oxford-street, W.

1871 King, Robert, M.B., Assistant Physician to the Middlesex Hospital, 48, Harley-street, W.

1852 Kingdon, J. Abernethy, Surgeon to the City Dispensary, and to the City of London Truss Society, 2, New Bank-buildings, Lothbury, E.C.

‡1856 Kingsley, Henry, M.D., Physician to the Stratford Infirmary, Stratford-on-Avon, Warwickshire.

1875 Koch, Edwin Lawson, M.D., Principal of the Medical School of Ceylon, Colombo, Ceylon. [Agents: Messrs. Henry S. King & Co., 65, Cornhill.]

1875 Lacy, C. S. De Lacy, St. George's Hospital.

‡1865 Lanchester, Henry Thomas, M.D., 53, High-street, Croydon.

1851 Langmore, John C., M.B., 20, Oxford-terrace, Hyde-park, W. (C. 1858-61.)

Elected

1865 Langton, John, Assistant Surgeon to St. Bartholomew's Hospital, 2, Harley-street, Cavendish-square, W.

1869 Larcher, O., M.D., Par., Laureate of the Institute of France, of the Medical Faculty and Academy of Paris, 97, Rue de Passy, Paris. [M. Kliensieck, Libraire, Rue de Lille, 11, Paris, per Messrs. Longman.]

1873 Latham, Peter Walkwork, M.D., Physician to Addenbrooke Hospital, and Downing Professor of Medicine, Cambridge University, 17, Trumpington-street, Cambridge.

1853 Lawrence, Henry John Hughes, Surgeon, Grenadier Guards' Hospital, Rochester-row, Westminster, S.W. (C. 1873-5.)

1859 Lawson, George, Surgeon to the Middlesex Hospital, and Surgeon to the Royal London Ophthalmic Hospital, Moorfields, 12, Harley-street, Cavendish-square, W. (C. 1870-1.)

1865 Leach, Harry (C.), Medical Officer of Health for the Port of London, Greenwich; 12, Rutland Park Villas, Catford Bridge, S.E. (C. 1876.)

1857 Leared, Arthur, M.D., Physician to the Great Northern Hospital, 12, Old Burlington-street, W. (C. 1874-5.)

1875 Lediard, Henry Ambrose, M.D., Medical Superintendent to Cleveland-street Asylum, 42, Cleveland-street, Fitzroy-square.

1852 Lee, Henry, (V.-P.), Surgeon to St. George's Hospital, 9, Savile-row, W. (C. 1860-2. V.-P. 1875-6.)

1867 Lees, Joseph, M.D., 112, Walworth-road, S.E.

1868 Legg, John Wickham, M.D., Physician to the Casualty Department, and Demonstrator of Morbid Anatomy, St. Bartholomew's Hospital, 47, Green-street, Park-lane, W. (C. 1874-5.)

1852 Leggatt, Alfred, 13, William-street, Lowndes-square, S.W. (C. 1866-7.)

‡1867 Leudet, T. Emile, M.D., Par., Professor of Clinical Medicine, 49, Boulevard Cauchoise, Rouen, France. [M. Kliensieck, Libraire, Rue de Lille, 11, Paris, per Messrs. Longman.]

1861 Lichtenberg, George, M.D., 47, Finsbury-square, E.C.

1875 Lingard, Alfred, St. Thomas's Hospital.

1848 Little, William John, M.D., 18, Park-street, Grosvenor-square, W. (C. 1851-2. V.-P. 1856-9.)

†1862 Little, Louis S., China. [18, Park-street.]

1874 Liveing, Edward, M.D., 52, Queen Anne-street, Cavendish-square, W.

1863 Liveing, Robert, M.D. (C.), 11, Manchester-square, W. (C. 1876.)

1873 Lucas, R. Clement, M.B., Assistant Surgeon to Guy's Hospital, 4, St. Thomas's-street, S.E.

1873 Lucey, William C., M.D., Ben Rhydding, by Leeds.

1876 Lyell, Robert Wishart, M.D., Surgical Registrar at the Middlesex Hospital, Headley Lodge, Croydon-road, Penge.

1871 McCarthy, Jeremiah, M.A., Surgeon to the London Hospital, 26, Finsbury-square, E.C.

Elected

1873 McConnell, J. F., Professor of Pathology, Medical College, Calcutta. [Per Grindlay & Co., Parliament-street.]

1871 MacCormac, William, Surgeon to St. Thomas's Hospital, 13, Harley-street, W.

1876 MacGrath, H. M., L.Q.K.C.P.I., 21, Colville-terrace East, W.

1858 Mackay, Allan Douglas, M.B., Stony Stratford, Bucks.

1875 Mackellar, Alexander Oberlin, Assistant Surgeon, St. Thomas's Hospital, Albert Embankment, S.E.

1873 Mackellar, Peter H., M.B., Medical Officer, Fever Hospital, Stockwell, S.W.

1870 Mackenzie, George Welland, 15, Hans-place, Sloane-street, S.W.

1870 Mackenzie, John T., Bombay, India. [East India United Service Club, 14, St. James's-square.]

1864 Mackenzie, Morell, M.D., Physician to the Hospital for Diseases of the Throat, and Lecturer on Diseases of the Throat at the London Hospital, 19, Harley-street, Cavendish-square, W.

1865 MacLaurin, H. N., M.D.

1876 Macnamara, Charles, Surgeon to the Westminster Hospital, 13, Grosvenor-street, W.

1875 Mahomed, Frederick Akbar, M.D., St. Mary's Hospital; 31, Lower Seymour-street, W.

1876 Maples, Reginald, St. Thomas's Hospital.

1857 Marcet, William, M.D., F.R.S., Villa Bianca, Cannes. (C. 1869-71.)

1868 Marsh, F. Howard (C.), Assistant Surgeon to the Hospital for Sick Children, Assistant Surgeon to St. Bartholomew's Hospital, 36, Bruton-street, Berkeley-square. (C. 1876.)

1876 Marshall, Francis John, St. George's Hospital.

1846 Marshall, John, F.R.S., Surgeon to University College Hospital, 10, Savile-row, W. (C. 1861.)

1856 Martin, Robert, M.D., 51, Queen Anne-street, Cavendish-square, W. (C. 1871-2.)

1852 Martyn, Samuel, M.D., Physician to the Bristol General Hospital, 8, Buckingham-villas, Clifton, Bristol.

1860 Mason, Francis, Assistant Surgeon to St. Thomas's Hospital, 5, Brook-street, Grosvenor-square, W. (C. 1873-5.)

1867 Mason, Philip Brookes, Burton-on-Trent.

†1858 Maunder, Charles F., Surgeon to the London Hospital, 16, Queen Anne-street, Cavendish-square, W. (C. 1869-71.)

†1852 May, George, Jun., M.B., Surgeon, Royal Berkshire Hospital, Reading.

1874 Meredith, William Appleton, M.B., 14, Old Burlington-street, W.

1859 Messer, John Cockburn, M.D., Assistant Surgeon, R.N., Her Majesty's Ship "Edinburgh," Queensferry, N.B.

‡1867 Mickley, Arthur George, M.B., Derby-road, Nottingham.

1866 Mickley, George, M.A., M.B., St. Luke's Hospital, Old-street, E.C.

†1859 Montefiore, Nathaniel, 36, Hyde-park-gardens, W.

1861 Morehead, Charles, M.D., 11, North Manor-place, Edinburgh.

Elected

1847 Morgan, John, 3, Sussex-place, Hyde-park-gardens, W. (C. 1856-8.)

1875 Morgan, John H., 12, Chapel-street, Grosvenor-square.

1874 Morison, Alexander, M.B., C.M., 70, Marquess-road, Canonbury, N.

1869 Morris, Henry, M.A., M.B., Assistant Surgeon to, and Lecturer on Practical Surgery at, the Middlesex Hospital, 2, Mansfield-street, Portland-place, W.

1875 Morton, John, M.B., Guildford.

1860 Moxon, Walter, M.D. (V.P.), Physician to Guy's Hospital, 6, Finsbury-circus, E.C. (C. 1868-70, V.P. 1876.)

1854 Murchison, Charles, M.D., LL.D. Edinb., F.R.S. (Treasurer), Physician to, and Lecturer on Medicine at, St. Thomas's Hospital, and Consulting Physician to the London Fever Hospital, 79, Wimpole-street, W. (C. 1859-62. S. 1865-8. T. 1869-76.)

1872 Murray, J. Jardine, 99, Montpellier-road, Brighton.

1864 Myers, Arthur B. R., Surgeon to 1st Battalion Coldstream Guards, Hospital, Vincent-square, Westminster, S.W. (C. 1872-3.)

1874 Nankivell, Arthur Wolcot, St. Bartholomew's Hospital, Chatham.

1873 Nettleship, Edward, 4, Wimpole-street, Cavendish-square, W.

1875 Newby, Charles Henry, Surgical Registrar, St. Thomas's Hospital.

1865 Newman, William, M.D., Stamford, Lincolnshire.

1868 Nicholls, James, M.D., Chelmsford, Essex.

1865 Nicoll, Charles R., M.D., Resident Medical Officer to the Charter House, 17, Charterhouse-square, E.C. (C. 1872-3.)

1864 Norton, Arthur T., Assistant Surgeon to St. Mary's Hospital, 6, Wimpole-street, Cavendish-square, W.

1856 Nunn, Thomas William, Senior Surgeon to the Middlesex Hospital, 8, Stratford-place, Oxford-street, W. (C. 1864-6.)

1871 Nunneley, Frederick Barham, M.D., Mickleover, Derbyshire.

1873 O'Farrell, George Plunkett, M.B., Tangier House, Boyle, Ireland; New Traveller's Club, 15, George-street, Hanover-square.

1850 Ogle, John W., M.D., Physician to St. George's Hospital, 30, Cavendish-square, W. (C. 1855-6. S. 1857-60. C. 1861-3. V.-P. 1865-8.)

1876 Oliver, John Ferens, M.D., 12, Old Elvet, Durham.

1860 Orange, William, M.D., Broadmoor, Wokingham, Berkshire.

1875 Ord, William Miller, M.B., Assistant Physician and Lecturer on Physiology at St. Thomas's Hospital, 7, Brook-street, Hanover-square, W.

1875 Osborn, Samuel, 17, Gresham Park, S.E.

1874 Owen, Charles William, 66, Kennington-road, Lambeth.

1865 Owles, James Allden, M.D., 204, Burlington-street, Liverpool.

1875 Page, Herbert William, M.B., M.C., Assistant Surgeon to St. Mary's Hospital, 28, New Cavendish-street, W.

Elected

1870 Paget, Sir James, Bart., D.C.L., F.R.S., Consulting Surgeon to St. Bartholomew's Hospital, 1, Harewood-place, Hanover-square, W.

1872 Parker, Robert William, S, Old Cavendish-street, W.

1874 Parker, Rushton, M.B., B.S., 65, Rodney-street, Liverpool.

1853 Parkinson, George, 50, Brook-street, Grosvenor-square, W.

1865 Pavy, Frederick William, M.D., F.R.S., Physician to Guy's Hospital, 35, Grosvenor-street, W. (C. 1872-4.)

1868 Payne, Joseph Frank, B.A., M.B., Assistant Physician to St. Thomas's Hospital, 6, Savile-row, W. (C. 1873-5.)

O.M. Peacock, Thomas Bevill, M.D. (Trustee), Physician to St. Thomas's Hospital, and Physician to the City of London Hospital for Diseases of the Chest, 20, Finsbury-Circus, E.C. (C. 1846-9. S. 1850-1. V.-P. 1852-6. C. 1858-61. *Pres.* 1865-6. V.-P. 1867-70.)

1872 Pearce, Joseph Chaning, M.B., C.M., The Manor House, Brixton-rise, S.W.

1863 Pearson, David R., M.D., 23, Upper Phillimore-place, Kensington, W.

1871 Phillips, Charles Douglas F., M.D., 107, Lancaster-gate, W.

1875 Philpot, Harvey John, Cresingham House, Peckham Rye, S.E.

1863 Pick, Thomas Pickering, Assistant Surgeon to, and Lecturer on Anatomy at, St. George's Hospital, 7, South Eaton-place, S.W. (C. 1870-1.)

1875 Pitman, Henry A., M.D., Consulting Physician to St. George's Hospital, 28, Gordon-square, W.C.

1867 Pitt, Edward G., M.D., 1, Cowley-villas, Leytonstone.

1876 Pitts, Bernard, M.A., M.B., Tharning Rectory, Oundle, Huntingdonshire.

1862 Pollock, Arthur Julius, M.D., Physician to Charing Cross Hospital, 85, Harley-street, Cavendish-square, W. (C. 1874-5.)

1846 Pollock, George D. (President), Surgeon to St. George's Hospital, 36, Grosvenor-street, W. (S. 1850-3. S. 1854-6. V.-P. 1863-5. P. 1875-6.)

1850 Pollock, James Edward, M.D., Physician to the Hospital for Consumption and Diseases of the Chest, Brompton, 52, Upper Brook-street, W. (C. 1862-4.)

1870 Poore, George Vivian, M.B., Assistant Physician to University College Hospital, 30, Wimpole-street, W.

1876 Port, Heinrich, M.D., 10, Finsbury-place North.

1854 Potts, William, 12, North Audley-street, Grosvenor-street, W. (C. 1870-2.)

1866 Powell, Richard Douglas, M.D., Assistant Physician to the Hospital for Consumption, Brompton, Assistant Physician to Charing Cross Hospital, 15, Henrietta-street, Cavendish-square, W. (C. 1873-5.)

1865 Power, Henry (C.), Ophthalmic Surgeon to St. Bartholomew's Hospital, 37a, Great Cumberland-place, Hyde-park, W. (C. 1876.)

Elected

1856 Priestley, William Overend, M.D., Consulting Physician-Accoucheur to King's College Hospital, and to the St. Marylebone Infirmary, 17, Hertford-street, Mayfair, W.

†1848 Purnell, John James, Surgeon to the Royal General Dispensary, Woodlands, Streatham-hill, S.W. (C. 1858-61.)

1865 Pye-Smith, Philip Henry, M.D. (C.), Assistant Physician to Guy's Hospital, 56, Harley-street, Cavendish-square, W. (C. 1874-6.)

O.M. Quain, Richard, M.D., F.R.S. (Trustee), Consulting Physician to the Hospital for Consumption and Diseases of the Chest, Brompton, 67, Harley-street, Cavendish-square, W. (C. 1846-51. S. 1852-6. T. 1857-68. *Pres.* 1869-70. V.-P. 1871-3.)

1859 Radcliffe, Charles Bland, M.D., Physician to the Westminster Hospital, 25, Cavendish-square, W.

1872 Ralfe, Charles Henry, M.D., M.A., Physician to the Seamen's Hospital, 26, Queen Anne-street, W.

1857 Ramskill, J. Spence, M.D., Physician to the London Hospital, Physician to the National Hospital for the Paralysed and Epileptic, 5, St. Helen's-place, Bishopsgate-street, E.C.

1848 Randall, John, M.D., Medical Officer, St. Marylebone Infirmary, 35, Nottingham-place, W. (C. 1864-6.)

1875 Ranger, W. G., 4, Finsbury-square.

1857 Ranke, Henry, M.D., Munich.

1865 Rasch, Adolphus A., M.D., Physician for Diseases of Women to the German Hospital, 7, South-street, Finsbury-square, E.C.

1870 Ray, Edward Reynolds, Dulwich, S.E.

1871 Rayner, Henry, M.D., Medical Superintendent, Middlesex County Lunatic Asylum, Hanwell, W.

1858 Reed, Frederick George, M.D., 46, Hertford-street, Mayfair, W.

1866 Reeves, Henry Albert, Assistant Surgeon to the London Hospital, 27a, Finsbury-square, E.C.

1875 Reid, Frank, Almond House, Upper Edmonton.

1875 Reid, Robert William, M.B., St. Thomas's Hospital.

1854 Reynolds, J. Russell, M.D., F.R.S., Physician to University College Hospital, 38, Grosvenor-street, W. (C. 1868-9.)

1871 Richards, J. Peeke, Medical Superintendent, Middlesex County Lunatic Asylum, Hanwell, W.

1866 Rivington, Walter, M.S. Lond., Surgeon to the London Hospital, 22, Finsbury-square, E.C.

‡1865 Roberts, David Lloyd, M.D., Physician to St. Mary's Hospital, Manchester, 23, St. John's-street, Manchester.

Elected

1871 Roberts, Frederick Thomas, M.D., 53, Harley-street, Cavendish-square, W.

1859 Robinson, Frederick, M.D., Surgeon-Major, Scots Fusilier Guards, 47, Claverton-terrace, St. George's-road, S.W. (C. 1871-3.)

1856 Robinson, Thomas, M.D., 35, Lamb's Conduit-street, W.C.

1865 Rogers, George Henry, 14, Old Burlington-street, W.

1858 Rolleston, George, M.D., F.R.S., Linacre Professor of Anatomy, University of Oxford, Park Grange, Oxford.

1876 Roper, Arthur, 17, Granville Park, Blackheath.

1858 Rose, Henry Cooper, M.D., Surgeon to the Hampstead Dispensary, High-street, Hampstead. (C. 1873-4.)

1876 Rose, William, M.B., B.S., Assistant Surgeon to King's College, 13, Old Cavendish-street.

1875 Rossiter, George Frederick, St. Thomas's Hospital.

1858 Rouse, James, Surgeon to St. George's Hospital, 2, Wilton-street, Grosvenor-place, S.W.

1869 Rutherford, William, M.D., F.R.S., Professor of Physiology in the University of Edinburgh.

1853 Salter, James A., M.B., F.R.S., Dental Surgeon to Guy's Hospital, 17, New Broad-street, City, E.C. (C. 1861-3.)

1852 Sanderson, Hugh James, M.D., 26, Upper Berkeley-street, Portman-square, W.

1854 Sanderson, John Burdon, M.D., F.R.S., Jodrell Professor of Human Physiology at University College, 49, Queen Anne-street, Cavendish-square, W. (C. 1864-7. V.-P. 1873-4.)

1875 Sangster, Charles, 15, Lambeth-terrace, S.E.

‡1847 Sankey, W. H. Octavius, M.D., Sandywell-park, near Cheltenham. (C. 1855.)

1871 Saunders, Charles Edward, M.D., 21, Lower Seymour-street, Portman-square, W.

1873 Savage, George Henry, M.D., Lecturer on Mental Diseases at Guy's Hospital, Bethlem Royal Hospital, St. George's-road, S.E.

1854 Scott, John, 49, Harley-street, Cavendish-square, W.

‡1847 Seaton, Edward C., M.D., Medical Officer of the Local Government Board, Rochester House, Surbiton. (C. 1859-61.)

1852 Semple, Robert Hunter, M.D., Physician to the Bloomsbury Dispensary, 8, Torrington-square, W.C. (C. 1859-61.)

1872 Sergeant, Edward, Medical Officer of Health, Bolton.

1856 Shillitoe, Buxton, Surgeon to the Great Northern Hospital, and to the Lock Hospital, 34, Finsbury-circus, E.C.

1855 Sibley, Septimus W., 4, Savile Row, W. (C. 1863-5.)

1875 Siddall, Joseph Bower, M.D., C.M., Bristol General Hospital, Bristol.

1847 Sieveking, Edward H., M.D., Physician to St. Mary's Hospital, 17, Manchester-square, W. (C. 1854-7. V.-P. 1864-5.)

Elected

O.M. Simon, John, D.C.L., F.R.S., Surgeon to St. Thomas's Hospital, 40, Kensington-square, W. (C. 1846-8. V.-P. 1855-9. *Pres.* 1867-8. V.-P. 1869-71.)

1866 Sims, Francis Manley Boldero, Assistant Surgeon to the Hospital for Diseases of the Skin, and Surgeon to the St. George's Dispensary, 25, Half-moon-street, Piccadilly, W.

1865 Sims, J. Marion, M.D., 267, Madison-avenue, New York.

1875 Smee, Alfred Hutchinson, 7, Finsbury-circus.

1872 Smith, Gilbart, M.B., Physician to the Royal Hospital for Diseases of the Chest, City-road, Visiting Physician to the Margaret-street Infirmary for Consumption, 68, Harley-street, Cavendish-square, W.

1875 Smith, George John Malcolm, M.B., Westminster Hospital.

1863 Smith, Henry, Surgeon to King's College Hospital, 82, Wimpole-street, Cavendish-square, W. (C. 1873-4.)

1866 Smith, Heywood, M.D., Physician to the Hospital for Women, 2, Portugal-street, Grosvenor-square, W.

Smith (P. H. Pye), see Pye-Smith.

1846 Smith, Protheroe, M.D., Physician to the Hospital for Women, 42, Park-street, Grosvenor-square, W.

1873 Smith, Richard T., M.D., Physician to the St. Pancras Dispensary, 53, Haverstock-hill, N.W.

1869 Smith, Robert Shingleton, M.D., Lecturer on Physiology, Bristol Medical School, 1, Leicester-place, Rokeby House, Clifton, Bristol.

1856 Smith, Spencer, Surgeon to St. Mary's Hospital, 9, Queen Anne-street, Cavendish-square, W.

1856 Smith, Thomas, Surgeon to St. Bartholomew's Hospital, 5, Stratford-place, Oxford-street, W. (C. 1867-9.)

1866 Smith, William, Melbourne, Australia.

1870 Smith, William Johnson, Surgeon, Seamen's Hospital, Greenwich, S.E.

1869 Smith, William Wilberforce, M.D., 2, Eastbourne-terrace, Bishop's-road, W.

1870 Snow, William Vicary, M.D., Richmond Gardens, Bournemouth.

1868 Southey, Reginald, M.D., Physician to St. Bartholomew's Hospital, 6, Harley-street, Cavendish-square, W.

1873 Sparks, Edward Isaac, M.B. [abroad].

1868 Spry, G. Frederick Hume, M.D., 2nd Life Guards, Army and Navy Club, Pall-mall, S.W.

1855 Squire, William, M.D., 6, Orchard-street, Portman-square, W. (C. 1870-2.)

1861 Squire, Alexander Balmanno, 24, Weymouth-street, Portland-place, W.

1876 Startin, James, 19, Sackville-street, Piccadilly, W.

1854 Stewart, William Edward, 16, Harley-street, Cavendish-square, W.

Elected

†1853 STREATFEILD, J. F., Surgeon to the Royal London Ophthalmic Hospital, Moorfields, and Ophthalmic Surgeon to University College Hospital, 15, Upper Brook-street, W.

1875 STURGE, W. A., M.B., 2, Patshall-place, Lawford-road, Kentish-town.

1863 STURGES, OCTAVIUS, M.D., Physician to the Westminster Hospital, 85, Wimpole-street, W.

1871 SUTHERLAND, HENRY, M.D., 6, Richmond-terrace, Whitehall, S.W.

1876 SUTRO, SIGISMUND, M.D., Senior Physician to the German Hospital, 37A, Finsbury-square.

1864 SUTTON, HENRY G., M.B. (C.), Physician to the London Hospital, Physician to the City of London Hospital for Diseases of the Chest, 9, Finsbury-square, E.C. (C. 1875-6.)

‡1867 SWAIN, WILLIAM PAUL, 20, Ker-street, Devonport.

‡1857 SYMONDS, FREDERICK, Surgeon to the Radcliffe Infirmary, 35, Beaumont-street, Oxford.

1870 TAIT, ROBERT LAWSON, Surgeon to the Birmingham and Midland Hospital for Women, 7, Great Charles-street, Birmingham.

‡1856 TAPP, W. DENNING, Hillside-house, Hatherley-road, Cheltenham.

1864 TATHAM, JOHN, M.D., 1, Wilton-place, Knightsbridge, S.W.

1870 TAY, WAREN, Surgeon to, and Demonstrator of Practical Anatomy at, the London Hospital, 4, Finsbury-square, E.C.

1871 TAYLOR, FREDERICK, M.D., Assistant Physician to Guy's Hospital, 15, St. Thomas's-street, S.E.

1861 TEEVAN, WILLIAM FREDERIC, Surgeon to the West London Hospital, 10, Portman-square, W.

1870 THOMAS, JOHN DAVIES, M.B., University College Hospital (India).

1852 THOMPSON, SIR HENRY, Knt., Emeritus Professor of Clinical Surgery in University College, 35, Wimpole-street, Cavendish-square, W. (S. 1859-63. C. 1865-67. V.-P. 1868-70.)

1874 THORNTON, JOHN KNOWSLEY, M.B., 83, Park-street, Grosvenor-square.

1872 THORNTON, WILLIAM PUGIN, Surgeon to the Hospital for Diseases of the Throat, and to the St. Marylebone General Dispensary, 42, Devonshire-street, Portland-place, W.

1865 THOROWGOOD, J. C., M.D. (C.), Lecturer on Materia Medica at the Middlesex Hospital, Physician to the City of London Hospital for Diseases of the Chest, 61, Welbeck-street, W. (C. 1876.)

1856 TOMES, J., F.R.S., Consulting Dental Surgeon to the Middlesex Hospital, 37, Cavendish-square, W. (C. 1867-9.)

1864 TONGE, MORRIS, M.D., Harrow-on-the-hill, Middlesex,

1872 TOWNSEND, THOMAS SUTTON, 68, Queen's Gate, South Kensington.

1876 TREDENNICK, WILLIAM MAGEE, M.D., 51, Warwick-road, South Kensington.

Elected

1851 Trotter, John W., Surgeon-Major, Coldstream Guards, Hospital, Vincent-square, Westminster, S.W., and the Tower. (C. 1865-9.)

1859 Truman, Edwin Thomas, Surgeon-Dentist in Ordinary to Her Majesty's Household, 23, Old Burlington-street, W.

1867 Tuckwell, Henry Matthews, M.D., Physician to the Radcliffe Infirmary, 64, High-street, Oxford.

1858 Tudor, John, Dorchester, Dorset.

†1875 Turner, Francis Charlewood, M.D., Assistant Physician to the London Hospital, 15, Finsbury-square, E.C.

1863 Turner, James Smith, Dental Surgeon to the Middlesex Hospital, 12, George-street, Hanover-square, W.

1858 Turtle, Frederick, Clifton Lodge, Woodford, Essex.

1854 Vasey, Charles, Surgeon-Dentist to St. George's Hospital, 5, Cavendish-place, Cavendish-square, W.

1867 Venning, Edgcombe, Assistant Surgeon, 1st Life Guards, Knightsbridge Barracks, 87, Sloane-street.

1875 Verdon, Walter, 252, Kennington-park-road, S.E.

1865 Vernon, Bowater John, Ophthalmic Surgeon to St. Bartholomew's Hospital, 43, Weymouth-street, Portland-place, W.

1868 Vincent, Osman, Surgeon to the Great Northern Hospital, 45, Seymour-street, Portman-square, W.

†1867 Wagstaffe, William Warwick, B.A. (Hon. Secretary), Assistant Surgeon to St. Thomas's Hospital, 2, Palace-road, Albert Embankment, S.E. (C. 1874. S. 1875-6.)

O.M. Waite, Charles D., M.D., Senior Physician to the Westminster General Dispensary, 3, Old Burlington-street, W.

1873 Walsham, William J., M.B., C.M., 27, Weymouth-street, Portland-place.

1859 Walters, John, M.B, Reigate, Surrey.

1847 Ward, T. Ogier, M.D., 12, Place de la Mare, Caen. (C. 1851-3.)

1858 Wardell, John Richard, M.D., Calverley-park, Tunbridge Wells.

1855 Watson, Sir Thomas, Bart., M.D., F.R.S., 16, Henrietta-street, Cavendish-square, W. (*Pres.* 1857-8. V.-P. 1859-63.)

1865 Watson, W. Spencer (C.), Surgeon to the Great Northern Hospital, Surgeon to the Royal South London Ophthalmic and to the Central London Ophthalmic Hospitals, 7, Henrietta-street, Cavendish-square, W. (C. 1875-6.)

1860 Way, John, M.D., 4, Eaton-square, S.W. (C. 1873-4.)

†1858 Weber, Hermann, M.D., Physician to the German Hospital, 10, Grosvenor-street, Grosvenor-square, W. (C. 1867-70.)

Elected

1876 Weir, Archibald, M.D., St. Mungho's, Great Malvern.

1864 Welch, Thomas Davies, M.D., Wyndham House, Ryde, Isle of Wight.

1861 Wells, John Soelberg, Ophthalmic Surgeon to King's College Hospital, and Surgeon to the Royal London Ophthalmic Hospital, 16, Savile-row, W.

1853 Wells, Thomas Spencer (V.-P.), Surgeon to the Samaritan Free Hospital for Women and Children, 3, Upper Grosvenor Street, W. (C. 1865-8. V.-P. 1876.)

†1851 West, Charles, M.D., Consulting Physician to the Hospital for Sick Children, 61, Wimpole-street, Cavendish-square, W. (C. 1856-7.)

1867 Whipham, Thomas Tillyer, M.B., Assistant Physician to St. George's Hospital, 37, Green-street, Grosvenor-square, W.

1869 Whipple, John H. C., M.D., Assistant Surgeon, 1st Battalion Coldstream Guards, Hospital, Vincent-square, Westminster, S.W.

‡1868 Whitehead, Walter, 248, Oxford-road, Manchester.

1870 Wicksteed, Francis William, Field House, Walthamstow, Essex.

1867 Wilcox, Richard Wilson, Temple-square, Aylesbury, Bucks.

1869 Wilkin, John F., M.D., M.C., New Beckenham, Kent.

1871 Wilkinson, J. Sebastian, Surgeon to the Central London Ophthalmic Hospital, 83, Wimpole-street, W.

1864 Wilks, Alfred G. P., M.A., M.B., Charlemont House, Spencer-road, Ryde, Isle of Wight.

1855 Wilks, Samuel, M.D., F.R.S., Physician to Guy's Hospital, 77, Grosvenor-street, W. (C. 1857-60. V.-P. 1869-72.)

1869 Williams, Albert, M.B., 4, York-terrace, Dartmouth-road, Sydenham, S.E.

O.M. Williams, C. J. B., M.D., F.R.S., Consulting Physician to the Hospital for Consumption and Diseases of the Chest, Brompton [47, Upper Brook-street, Grosvenor-square, W.]. (*Pres.* 1846-47. V.-P. 1848-52. C. 1853-55. V.-P. 1858-61.)

‡1858 Williams, Charles, Assistant Surgeon to the Norfolk and Norwich Hospital, 9, Prince of Wales-road, Norwich.

1866 Williams, Charles Theodore, M.B. (C.), Physician to the Hospital for Consumption and Diseases of the Chest, Brompton, 47, Upper Brook-street, Grosvenor-square, W. (C. 1875-6.)

1872 Williams, John, M.D., Assistant Obstetric Physician to University College Hospital, 28, Harley-street, Cavendish-square, W.

1864 Williams, W. Rhys, M.D., Bethlem Royal Hospital, Lambeth-road S.E.

1863 Willis, Francis, M.D., Bracebrough, Stamford.

1859 Wilson, Edward Thomas, M.B., Montpelier-terrace, Cheltenham.

1859 Wilson, Robert James, F.R.C.P. Ed., 7, Warrior-square, St. Leonard's-on-Sea.

1863 Wiltshire, Alfred, M.D., Joint Lecturer on Midwifery at St. Mary's Hospital, 57, Wimpole-street, Cavendish-square, W.

Elected

‡1861 Windsor, Thomas, Surgeon to the Salford Royal Hospital, 44, Ardwick-green, Manchester.

1874 Wiseman, John Greaves, Dearden-street, Ossett, Yorkshire.

1865 Witherby, William H., M.D., Pitt-place, Coombe, Croydon.

1850 Wood, John, F.R.S., Surgeon to King's College Hospital, 68, Wimpole-street, W. (C. 1857-59. V.-P. 1872-4.)

1854 Wood, William, M.D., Physician to St. Luke's Hospital, 99, Harley-street, W.

1876 Wood, William Edward Ramsden, M.A., 99, Harley-street, W.

1865 Workman, Charles John, M.D., Titherley, Teignmouth, Devon.

1863 Worley, William Charles, 1, New North-road, Hoxton, N.

1859 Wotton, William Gordon, King's Langley, Herts.

1852 Wright, Edward John, 169, Clapham-road, S.W.

1869 Wyman, W. S., M.D., Westlands, Upper Richmond-road, Putney, S.W.

1862 Yeo, J. Burney, M.D., Physician with Charge of Out-Patients to King's College Hospital, and Assistant Physician to the Brompton Hospital for Consumption, 44, Hertford-street, Mayfair, W.

1872 Young, Henry, M.B., Monte Video, South America.

ANNUAL REPORT OF COUNCIL.

1875-6.

The Council are glad to be able to congratulate the Members upon the continued success of the Society.

The number of members has now reached 545, and of these 35 have been added during the past year, a fact which may be taken to indicate that the Society has not diminished in public appreciation.

Four members have been removed by death during this period, viz. Messrs. J. B. Foster, J. D. Hill, S. W. Moore, and Dr. Charles E. Squarey, and two have resigned.

The ordinary meetings have been better attended than in any previous year, and the specimens exhibited have not diminished in interest or number.

The largeness of the number of specimens offered for exhibition has engaged the attention of the Council, and they have now under consideration certain suggestions for improving the management of the meetings.

During the past year a discussion was opened by Dr. Charlton Bastian upon the Germ Theory of Disease, which occupied three entire evenings. The remarks made in the course of the debate form a valuable epitome of the different views held by observers of different schools, and will, the Council trust, afford to pathologists a basis for future research in this obscure but important subject.

The Council desire to offer their thanks to Dr. Bastian and to those gentlemen who took part in the discussion.

The meetings were, as on the occasion of the previous debates, very numerously attended, and the Council, recognising their success, and bearing in mind the importance of recording the different views held upon general questions of pathological interest, propose to set apart certain evenings in the present session to the consideration of some important points in the pathology of Syphilis. They have much pleasure in congratulating the Society upon the fact that the discussion will be opened by Mr. Jonathan Hutchinson early in February.

The Morbid Growths Committee have continued their labours, and their Reports have added greatly to the accuracy and value of the descriptions of the specimens submitted to the Society. The Committee has suggested that it would add to the interest and value of their investigations if, from time to time, diseased tissues were submitted to them other than those which are commonly termed new growths.

The names of Mr. Butlin and Mr. Godlee have been added to the Committee.

During the past year the members have been presented with a copy of the Index to the 'Transactions' from Vol. XVI to XXV inclusive, through the generosity of Dr. Peacock, one of the former Presidents, and the Council cannot refrain from again expressing their warmest thanks to him, and to the donors of the first Index, for the very valuable means of reference which they now possess to the stores of pathological observations contained in their 'Transactions.'

The advisability of the President continuing in office for one year instead of two was forcibly urged by the President in his introductory address, but the Council are not prepared to adopt the suggestion upon the present occasion.

From the Treasurer's Balance Sheet, which is appended, it will be seen that the total amount invested in the names of the Trustees is £823 10*s.* 6*d.*, and the balance at the Bankers' £148 6*s.* The total receipts for the year have amounted to £508 15*s.* 9*d.*, of which £495 18*s.* 4*d.* have been expended, including £231 12*s.* 9*d.* the cost of the 'Transactions,' and the sum of £120 invested in three per cent. Consols.

It will be noticed that the amount received for the sale of the 'Transactions,' £57 16*s.* 7*d.*, is larger than ever before; and this, together with the increase in the number of members, indicates the advisability of printing a larger number of copies of the 'Transactions' in future.

GEORGE POLLOCK.

THE PATHOLOGICAL SOCIETY

In Account with the Treasurer, Dr. MURCHISON.

Dr.							Cr.					
To *Meetings*: 1875.	£	*s.*	*d.*	£	*s.*	*d.*	1875	£	*s.*	*d.*	£	*s.*	*d.*
Payment to Royal Medical and Chirurgical Society for use of Rooms, &c....	63	0	0				By Balance at Bankers', January, 1874				135	8	7
Refreshments, Waiters, and Management of Meetings	31	10	0				*Subscriptions* received:						
Green Shades and altering of Gas Lamps	0	9	6				354 Annual Subscriptions for 1874-75	371	14	0			
Expenses of Morbid Growths Committee	5	0	0				1 ditto, Arrear	1	1	0			
				99	19	6	30 Entrance Fees	31	10	0			
Petty Cash, per Hon. Secretaries	3	7	5				1 Composition Fee, Resident Member	15	15	0			
Ditto, per Mr. Wheatley, Postages, &c.	5	12	4				4 ditto, Non-Resident	8	8	0			
Cheque Book, per Union Bank	0	4	4								428	8	0
				9	4	1	'Transactions,' Sale of..				57	16	7
Stationery (Wodderspoon).....	11	0	6				Dividends received:						
Receipt Books (Odell)	0	17	0				On £692 18*s.* 3*d.*, January 5th, 1875	10	6	2			
				11	17	6	On £823 10*s.* 6*d.*, July 5th, 1875 ...	12	5	0			
Assistance to Secretaries, Treasurer, &c.:											22	11	2
Assistance to Secretaries (Wheatley)...	7	7	0										
Collection of Subscriptions and Accounts	14	7	6										
Posting Ledger (McDermot)	1	10	0										
				23	4	6							
Transactions: Expenses of Vol. XXVI (600 copies):													
Printing (Adlard).....	157	3	3										
Woodcuts (Butterworth and Heath)...	10	8	6										
Lithography (G. H. Ford)	36	15	0										
Ditto (Mintern)	24	3	0										
Composition of Index (Wheatley).....	3	3	0										
				231	12	9							
				375	18	4							
Purchase of £130 12*s.* 3*d.*, 3 per cent. Consols				120	0	0							
Balance in hand, carried down				148	6	0							
				£644	4	4					£644	4	4
							Balance brought down, at Bankers'				£148	6	0

Examined and found correct, December, 1875.

Auditors. { MARCUS BECK, SIDNEY COUPLAND.

LIST OF SPECIMENS AND REPORTS

BROUGHT BEFORE THE SOCIETY DURING THE SESSION 1875-6.

I.—DISEASES, ETC., OF THE NERVOUS SYSTEM.

II.—DISEASES, ETC., OF THE ORGANS OF RESPIRATION.

III.—DISEASES, ETC., OF THE ORGANS OF CIRCULATION.

IV.—DISEASES, ETC., OF THE ORGANS OF DIGESTION.

(A) TONGUE AND DIGESTIVE CANAL.

(b) DISEASES OF THE LIVER, PERITONEUM, ETC.

V.—DISEASES, ETC., OF THE GENITO-URINARY ORGANS.

(A) KIDNEYS, BLADDER, ETC.

(B) MALE GENITAL ORGANS.

(C) FEMALE GENITAL ORGANS.

VI.—DISEASES, ETC., OF THE OSSEOUS SYSTEM.

VII.—DISEASES, ETC., OF THE ORGANS OF SPECIAL SENSE.

VIII.—TUMOURS.

IX.—DISEASES, ETC., OF THE DUCTLESS GLANDS.

(A) DISEASES OF SPLEEN AND LYMPHATIC GLANDS.

(B) DISEASES OF SUPRA-RENAL CAPSULES.

(c) DISEASES OF THYROID AND THYMUS GLANDS.

X.—DISEASES, ETC., OF THE SKIN.

XI.—MISCELLANEOUS SPECIMENS.

REPORTS OF THE COMMITTEE ON MORBID GROWTHS.

LIST OF PLATES.

WOODCUTS.

REPORT.

SESSION 1875-76.

I.—DISEASES, ETC., OF THE NERVOUS SYSTEM.

1. *Subarachnoid hæmorrhage of spinal cord.*

By Thomas S. Dowse, M.D.

W. D—, æt. 50, admitted into Central London Sick Asylum, September 28th, and died September 30th, 1875. The history of this case is obscure, but as far as could be ascertained he had drank freely, yet his health was usually good. He was working one day in the sun (ten days before his death) and suddenly complained of great pain in the head, which was soon followed by giddiness, vomiting, shaking and sub-consciousness. Five days before his death he got up and felt so much better that he went out for a walk, but soon returned, had a fit with convulsions of the body generally, but mostly of the right side; he became comatose and died three days after.

The signs of hæmorrhage over the convolutions were well marked upon his admission, when the following note was made:—He lies in a state of coma, not profound. There is slight cerebral perceptive power. The special senses seem almost in abeyance, that of hearing being the most active. There is deep-seated right æsthesia, almost amounting to hyperæsthesia, all over the body. Respiration 30, pulse 120; temperature, R. $103\frac{3}{10}$° F.; L. $103\frac{3}{10}$° F. It is impossible to say how far the cranial nerves are involved; there is no palsy of the seventh. Reflex excitation of the facial muscles is easily induced, but more on the left than the right side. There is marked rigidity of both upper and lower limbs, the former are flexed, the latter extended, and

not only are they hyperæsthetic, but reflex movements are easily excited.

He appears to possess some voluntary power over the arms, but none over the legs. The urine was retained, but free from albumen. On the day of his death he became profoundly comatose. Respiration 44; pulse 160; temperature 106° F.

At the *post-mortem* the vessels of the dura mater were found engorged with blood, and blood was extravasated over the hemispheres and cerebellum. The anterior lobes were the only parts free. The hæmorrhage was more dense over the parieto-occipital and cerebellar lobes. (This is interesting in reference to the voluntary power which he possessed over the upper extremities.) At the base of the brain there was a mass of clotted blood extending from the anterior perforated space to the medulla. The vessels were not atheromatous, and the seat of hæmorrhage was not ascertained. When the cord was removed the same condition presented itself. The hæmorrhage extended between the arachnoid membrane and pia mater over the anterior and posterior surfaces alike.

October 18*th*, 1875.

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2. *Aneurysm of the anterior communicating artery of the brain; rupture; subarachnoid hæmorrhage.*

By W. S. Greenfield, M.D.

Israel S—, æt. 45, a bricklayer, was admitted into St. Thomas's Hospital, under the care of Mr. Simon, on May 31st, 1875, having fallen off a scaffold from a height of about thirteen feet. He was insensible on admission, but regained consciousness in a short time, and was found to be suffering from a fracture of the pelvis and a scalp wound on the left side of the head. On the following day he appeared to be doing well; there was no headache, and no paralytic symptoms are noted to have existed. He continued in much the same condition until June 8th, when he is said to have died suddenly in a fit, but there are no precise notes of its duration or symptoms.

Post-mortem, June 9th, at 2 p.m., twenty-one hours after death. Body of a strong, well-nourished man; rigor mortis well marked.

There was a scalp wound of triangular shape over the left parietal eminence, and beneath this there was slight hæmorrhage, but no fracture of the skull. On removing the calvarium it was found to be normal, and there was no trace of injury to the dura mater, and no hæmorrhage external to it. On cutting through the dura mater and removing it in the usual manner, no blood escaped, but on the upper surface of both hemispheres blood was seen extending upwards beneath the arachnoid over the occipital lobes and along the Sylvian fissures, and to a less degree over the frontal lobes. At the inferior aspect of the brain a small quantity of blood was found lying in the arachnoid cavity; this was entirely uncoagulated, and on removing the brain a large quantity of blood escaped. After removal the base of the brain was found to be completely covered by a mass of blood and clot, which surrounded the medulla and upper part of the cord, but which was almost entirely beneath the arachnoid. A layer of semi-coagulated blood completely concealed the parts at the base, and extended upwards along the fissures towards the vertex. The hæmorrhage was most marked around the pons and medulla, as if originating there, and there was a large quantity between the hemispheres of the cerebellum. There was no sign whatever of laceration or contusion of the brain in the neighbourhood.

On section of the hemispheres they were found to be normal, but the lateral ventricles contained some blood-stained fluid, and small coagula projected into the posterior part. There was a firm clot filling the fourth ventricle, and extending also along the iter a tertio ad quartum, which projected into the posterior part of the third ventricle through the transverse fissure, these coagula being continuous with the layer of blood surrounding the pons and upper part of the medulla. There was, however, no sign of injury to the brain substance anywhere. The vessels at the base were slightly atheromatous, especially the internal carotids, but there was no injury discoverable at this stage. The spinal cord was completely surrounded throughout its entire length with a layer of partially coagulated blood. The blood immediately covering the parts in the interpeduncular space was firmly coagulated, whilst that which extended more widely was only partially so. The brain was therefore subjected to a continuous stream of water for a day or two, and the vessels again examined with great care, with the following result:—

The internal carotids, middle and posterior cerebrals, with their branches, were found to be slightly atheromatous, but otherwise normal. The anterior cerebrals were also normal, but somewhat rigid and larger than usual. It was observed that they were surrounded by very firmly coagulated blood, and on tracing them forwards a small mass, apparently a clot, was found lying between them, just in front of the anterior communicating artery. On further examination this was found to be a small aneurismal sac, which projected from the superior and anterior aspect of the anterior communicating artery. The sac was the size of a small pea and for the most part filled with firmly coagulated clot. It was surrounded by a layer of fibrous tissue and was partially adherent at the sides to the two anterior cerebral arteries. At its origin from the vessel its wall was extremely thin, especially on the upper aspect, and at this point was a perforation about $\frac{1}{12}$th inch in diameter, close to its junction with the vessel. Owing to the situation of this opening the blood must have passed almost directly backwards in its escape, and its projection upwards prevented its being well seen from the under surface. The vessels were carefully dissected out and put aside to be preserved, but owing to some rough usage the small sac was torn off, leaving only the opening in the artery, and spoiling the specimen. The drawing had, however, been made beforehand. The drawing, made from the fresh specimen, shows the situation of the sac and the size and position of the orifice.

Remarks.—The facts revealed by the *post-mortem* seem to explain in a striking manner the symptoms and history of the case. In all probability the hæmorrhage occurred at three or four different periods, and the attacks of hæmorrhage were the cause of the fits. That the hæmorrhage did not all occur at one and the same time seems to be clearly shown by the very firm coagulation of the blood over the interpeduncular space, and the nearly fluid condition of that which was more widely diffused. Three distinct attacks appear to be indicated by the conditions found *post-mortem*, and it may be suggested that the suddenness of the patient's death was owing to the sudden pressure of the effused blood on the floor of the fourth ventricle. The bleeding, in fact, seems to have stopped at the moment when the fourth ventricle was filled, otherwise there was no evident reason why it should not have gone further and reached the lateral ventricles.

Pathologically the case is of interest from the extreme rarity of

the disease in this situation, only one case, so far as I have been able to discover, being on record, viz. one by Dr. McDowall, in the 'Lancet,' August 7th, 1875, in which, however, the aneurism produced no symptoms, and did not rupture, being discovered only after death. It is also of interest from the clearness with which the history of a case was traceable after death. The case is also not without value in its medico-legal relations, as it was a question whether the patient's death was due to the fall from the scaffold, and whether, if so, blame was to be attached to any one for its insecure condition. It is, of course, to some extent an open question whether the fall might have been the cause of the rupture of the aneurism, but it seems more probable, from the conditions found after death, that the man fell in a fit, which was due to the first rupture of the sac. Unfortunately, attention was not directed specially to the cerebral symptoms after admission to the hospital. There can be no doubt that the sudden death was due to the later rupture. Happily for the employers, the coroner's jury in their wisdom decided that the man died of heart disease (which did not exist), and that his death was in no way connected with the accident.

October 19*th*, 1875.

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3. *Tumour (? syphilitic) of left anterior cerebral artery, producing thrombosis and hemiplegia.*

By W. S. Greenfield, M.D.

J. T. G——., æt. 37, a seaman, was admitted to St. Thomas's Hospital, under the care of Dr. Stone, on August 13th, 1875. It was ascertained that he had had a syphilitic sore nine years before, when at Malta, but no other history was noted. When admitted he was suffering from incomplete hemiplegia, which he stated had come on suddenly a week before, when he fell down, but without loss of consciousness. There was some weakness of the left arm and leg, and the right leg was also weak, but there was no facial or lingual paralysis. Temperature normal; urine free from albumen. Treated with Pot. Iod. gr. x ter die. On August 23rd he was seized with a "fit," which appears to have been without loss of

consciousness, but vomiting and headache followed it, the speech was affected, and the left eye was said to be moved convulsively. On the following day only slight ptosis of the left eyelid remained, but there was no obvious affection of the ocular muscles. On August 27th repeated vomiting occurred, which ceased on the 28th, but the left eyelid drooped more and the left pupil seemed smaller than the right, although active. On the 30th he spoke indistinctly, and there was some loss of memory. On November 5th he became much worse, and the temperature rose. On the 6th he was only half-conscious, moving the right hand about, answering very indistinctly when spoken to; the ptosis of the left eye increasing. On the 7th the temperature rose to 105·8° F., and symptoms of pneumonia showed themselves. The patient gradually sank, the temperature continuing elevated. (For the clinical notes I am indebted to Dr. Stone.)

Post-mortem examination, twenty-five hours after death.—Body emaciated; a large, deeply pigmented scar over the front of the left leg.

Head.—Calvarium normal. Dura mater somewhat thick and opaque, but free from patches of thickening. Arachnoid normal. Much subarachnoid fluid. Pia mater normal. On section of the hemispheres in the usual manner the cortical grey matter appeared somewhat too vascular in its inner layers, especially over the anterior lobes; and the centra ovalia also showed some venous engorgement. The lateral ventricles contained a slight excess of fluid, which on the left side was somewhat turbid. The anterior part of the nucleus caudatus of the left corpus striatum was of a yellow colour, very soft to the touch, and readily washed away by water, the corresponding portion of the right being unaffected by a similar stream. This softening was found to affect solely the anterior extremity of the nucleus caudatus, and a thin layer of the adjacent white matter in front of and to its outer side. The remainder of the centres appeared normal. The *vessels* at the base were found to present numerous patches of thickening and rigidity, and also small atheromatous nodules, some of which considerably diminished the calibre of the vessels. On the middle cerebral artery, not far from its origin, was such a thickening, which did not, however, completely obstruct the vessels, and no sign of thrombosis in this situation could be discovered on close examination.

On the left anterior cerebral artery, close to but just behind the

anterior communicating artery, was a small rounded nodule, the size of a pea, measuring about six millimètres in diameter. This was closely adherent to the wall of the vessel, which, in fact, it appeared to surround, but projecting chiefly in an upward and forward direction, so as to press upon and be partially imbedded in the left optic nerve close to the chiasma. The nodule was of yellowish colour, slightly translucent, and extremely hard. From this point a firm thrombus extended backwards in the anterior cerebral artery to near its origin, but the middle cerebral was not involved. The middle cerebral was of small size, and a branch from the anterior cerebral supplied the anterior part of the corpus striatum, and was completely plugged.

The tumour could not be submitted to microscopic examination, as it was preserved for a museum specimen; its nature must therefore remain doubtful.

There was extensive confluent lobular pneumonia, but for the most part the other organs were healthy.

October 19*th*, 1875.

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4. *Hæmorrhage into pons Varolii and fourth ventricle.*

By Thomas S. Dowse, M.D.

Dr. Dowse showed the brain of a woman, æt. 66, where hæmorrhage had taken place into the pons Varolii and fourth ventricle. Her health had been excellent until about two years ago, when she became suddenly unconscious and remained so for three days. There was a complete hemiplegia of the left side of motion, and partially of sensation. From this time her mind became deranged and she suffered from epileptic seizures with convulsions of the right half of the body. The temperature of the left was usually two degrees higher than that of the right side.

On October 12th, 1875, she had what the nurse thought to be one of her fits. The right side was convulsed and at first there was marked right facial palsy, the head and eyes were drawn to the left and the pupils were minutely contracted, but in a very short time

profound coma set in, with stertorous breathing; she remained in this state for about twelve hours, when she died. From this it is evident that considerable hæmorrhage rapidly ploughed up the substance of the pons Varolii bilaterally, yet from the primary signs, evidenced by right facial palsy, the right half was the first to become involved. When the brain was removed considerable disease was at once evident. The vessels of the pia mater were engorged with blood, and subarachnoid hæmorrhage existed over both lateral lobes of the cerebellum, more markedly over right temporo-sphenoidal convolutions.

The substance of the left hemisphere was fairly healthy, but the right was much diseased. The centrum ovale showed two or three irregular cavities extending upwards to the grey matter of the anterior and posterior central convolutions; the most part of the brain substance here, as well as in the optic thalamus and crura on this side, had undergone a yellowish-brown staining from hæmorrhages of old date. The corpus striatum was not involved.

At the base, from the chiasma anteriorly to the medulla posteriorly, was a mass of extravasated blood, the result of the giving way of the left half of the pons Varolii, into which the hæmorrhage had first taken place. When the clot was removed from the surrounding brain matter a total destruction of the left half of the pons was at once evident. The right side appeared normal until a bit of its structure was removed. It was then seen that the hæmorrhage had invaded the entire substance of the pons Varolii. The fourth ventricle was full of blood. The arteries were atheromatous. The specimen has been examined to determine, if possible, the ruptured vessel, but it could not be detected. The hæmorrhage had completely destroyed the substance of the pons.

October 18*th*, 1875.

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5. *Glio-sarcoma of the brain.*

By Thomas S. Dowse, M.D.

J. W—, æt. 48, was admitted into the Central London District Asylum at Highgate, on the 13th April, 1875, with the follow-

ing history:—Is one of six children, all excitable. Two of his brothers are now under my care; one suffers from phthisis, the other from brain disease. Until five years ago he was a healthy man, but after fits of intemperance he would suffer from severe headaches and behave like a madman.

On Whit Tuesday, in the year 1873, after having drank very freely, he was for the first time seized with a fit and became unconscious for a few minutes. These fits continued at varying intervals for a week. The right side was much convulsed both during the fits and in the intervals. The right arm quite regained its normal tone, but not the leg, so that from this time the legs were weak, and his gait was peculiar.

On admission the intellect was clear, the memory defective, and when questioned he became confused; in fact, he seemed silly. The eyes were bright, the sight good, but upon ophthalmoscopic examination the discs were hazy. He complained occasionally of pain in the head, but not of giddiness. The cranial nerves were all unaffected. He was quite unable to stand erect without great effort, and he was also unable to balance himself; his movements were very similar to those of an amateur rope-walker. His whole bearing gave one the idea, not that he was wanting in volitional power, but that he lacked confidence in himself to regulate his power of movement. There was not the absence of voluntary co-ordinating power in the lower limbs which is common to sclerosis of the posterior columns of the cord, neither had he the increasing feebleness of the legs which is also so common with diffuse disease of the cerebellum. I rather surprised him by making him run round the ward within a month of his death without falling, yet it was impossible for him to walk three steps in a deliberate manner, so that his usual mode of progression was that of a jog-trot, with the body inclined slightly forwards.

On June 11th, more than two months after his admission, he had an epileptiform seizure, and from this time his condition changed. He had great difficulty in raising himself up in bed; there was loss of control over the sphincters; he was lethargic, but the cranial and bulbar nerves remained unaffected. There was still no paralysis of the upper extremities, but voluntary power in the lower limbs was very limited, and always associated with tremor. Cutaneous sense and sensibility were normal, but reflex movements were not usual. The localisation of the perception of touch was peculiar. For instance,

when I touched the right foot he acknowledged it correctly enough, but he also maintained that I was touching the right foot when I was touching some other part of the body. Yet when contact was withdrawn he knew it immediately. The urine was highly ammoniacal, but free from albumen.

From this until the hour of his death he became more prostrate, and died comatose, the temperature during the last part of his existence varying from 101° to 104° F.

Post-mortem examination, made twenty-four hours after death. —There was well-sustained cadaveric rigidity.

The heart, lungs, and abdominal viscera were healthy.

The spinal cord presented evidence of slight softening about the ninth dorsal region, and its pia mater was hyper-vascular.

The brain when removed and examined at the base gave no evidence of arterial change; neither were the nerves softened, but the substance of the left olfactory convolution projected downwards, evidently from some pressure above.

The membranes covering the grey matter, and the grey matter itself, were healthy, both here and over the convolutions of the hemispheres.

Upon section at a level with the corpus callosum it was found that considerable change had taken place, involving especially the anterior lobes, and not extending backwards beyond the post-median convolution. A growth was discovered, of a deep red colour and spongy texture, soft but yet tenacious and presenting here and there upon section small cretaceous and cheesy masses. I am inclined to think that it originated in the choroid plexus, possibly where it communicated with the lateral ventricles through the foramen of Monro. It had completely replaced the anterior part of the corpus callosum, and when the lateral ventricles were exposed, its outline was fairly traceable, and its invasion of the brain substance well defined. Its body was of cone-like form, the base replacing the anterior part of the corpus callosum, and resting upon the fornix, the apex of the cone being free and extending nearly to the pineal gland. It occupied the anterior horns of the lateral ventricles, and exercised considerable pressure upon the striate bodies, to which it had merely contracted slight adhesions. Both hemispheres were invaded—on the right side only the outer two thirds of the callosal convolution; on the left, it extended considerably into the centrum ovale from below upwards and from within outwards as far as the grey matter,

but not involving it. The choroid plexuses in the posterior horns had undergone fatty change.

Thus it will be seen that the grey matter of the hemispheres was not involved. Secondly, that the motor tract was not implicated, excepting by pressure upon the corpora striata, which is very important in reference to the man's power of locomotion. The corpus callosum had almost disappeared.

Upon microscopic examination the growth was found to be highly vascular. It consisted of round, oval, and spindle cells, some nucleated, others not, embedded in a finely fibrillated stroma. Here and there might be seen comparatively large polynucleated cells.

November 16*th*, 1875.

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6. *Gumma syphiliticum of posterior cerebral sinuses and tentorium cerebelli.*

By Thomas S. Dowse, M.D.

W. W.—æt. 39, was admitted into the Central London Sick Asylum, Highgate, on November 16th, 1874, and died November 1st, 1875. Family history of no importance. He contracted syphilis when a young man, but suffered little from it at that time. For many years the state of his health has been indifferent, but for the past eighteen months he has been unable to follow any occupation. He has never had a fit of any kind. It appears that he was compelled to give up work by reason of the severe shooting pains affecting the skull, but more particularly the frontal bones. The sight failed, and letters would appear double, straight lines looked curved and then ran into each other. The smell also was impaired, and offensive matter was discharged from the nostrils. The power of locomotion was interfered with ; he would feel giddy and stop short from attacks of *petit mal*. The bowels were obstinately confined and he vomited after nearly every meal. In addition, he suffered from otalgia, with intermittent otorrhœa.

When I first saw him the above signs and symptoms were well marked ; he was pale, and over the right frontal eminence was a cica-

trix the result of previous ulceration, from which some necrosed bone had made its escape. It prevented the due movement of the occipito-frontalis muscle and gave one the idea that the seventh nerve was involved, which was not the case. The ophthalmoscope showed hazy outline of optic discs, the veins large, the arteries small; in fact, a neuro-retinitis going on to atrophy. His gait was not unsteady, but he stopped short in walking, became confused, and would fall unless he caught hold of something to support himself. There was neither objective nor subjective paresis of the cranial or spinal nerve centres. The special senses of smell, sight, and hearing, were impaired from localised disease only. Cutaneous sensation and sensibility were everywhere normal, and the muscles responded readily to the galvanic current. The sphincters were unaffected. There were no formication, tremors, convulsive or reflex movements. He complained of the head feeling heavy, so that at times he was unable to raise it from the pillow. But one of the especial features of the case was the association of signs and symptoms, such as cessation of discharge from the ear, followed by intense headache, persistent vomiting and obstinate constipation. These attacks were paroxysmal and lasted for eight or ten days. At other times he was tolerably well. The intellect was rarely affected, but the memory was variable. The pulse, temperature, and respirations, were normal.

He died after an attack such as that just described, apparently from want of nervous power.

Post-mortem.—The cranium was unusually thick, compact and heavy, its outer surface was covered with nodular elevations. The inner surface of the skull was free from any outgrowths. The dura mater over the anterior lobes was normal, but on nearing the torcular Herophili the presence of some foreign growth became manifest. It was impossible to remove the brain free from the dura mater, on account of the adhesions between it and the cerebellar and occipital lobes, but more particularly between the upper surface of the left lateral lobe of the cerebellum and the under surface of the posterior convolutions of the parietal lobes of each side. It will be seen that the growth has invaded and occluded all the sinuses in relation with the torcular Herophili except the two occipital. It has also invaded the whole of the tentorium cerebelli, but more on the left than on the right side. When the straight sinus was divided the growth presented a yellowish look; it was of firm texture and not altogether adherent

to the wall of the vessel, for around its outer border was a zone of dark coloured blood.

Under the microscope it was seen to be made up of wavy connective tissue infiltrated with round and spheroidal cells.

November 16th, 1875.

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7. *Glioma of left cerebral hemisphere.*

By W. R. Gowers, M.D.

The specimen of glioma of brain is exhibited partly because it presents a typical example of the mode of growth of a glioma, but especially because the symptoms which it caused were of considerable interest, in their sudden onset and bilateral character.

G. W—, æt. 46, a commercial traveller, was admitted into University College Hospital, under the care of Sir William Jenner, on September 21st, 1875, suffering from right hemiplegia. He was an unmarried man, and had been a "hard drinker." No history or evidence of syphilis could be obtained. From the imperfect account of his early symptoms, which was received from his friends, it appeared that in the middle of July, after having seemed "stupid" for a day or two, he lost consciousness for a short time (probably a quarter of an hour). On recovery he was able to return home, walking some distance with the help of a boy. On reaching home weakness of the right side was observed and some difficulty in expression. These symptoms continued and difficulty of movement was noticed over and above the weakness. When he wanted to put a cup down he would put out his arm and put the cup down a foot further off than he intended. So also with the leg in stepping. No inequality in the face was noticed. The difficulty in expression increased so that often he was quite unable to say what he wished. When by his gestures his friends guessed what he wanted to say and expressed it, he would answer correctly "Yes" or "No," as the case might be. He had one or two slight attacks of loss of consciousness, but without any rigidity of limb.

On September 17th, having been alone for some time, he was found unconscious on the floor. The right arm and leg were power-

less. He recovered partial consciousness, but took little notice, and did not speak. In this state he continued until he was brought to the hospital four days subsequently.

On admission he did not speak, even in answer to questions, but he appeared to understand what was said to him. The movements of the eyeballs were perfect; there was slight paralysis in the lower part of the face on the right side. The tongue deviated to the right. There was complete paralysis of the right arm and all but complete paralysis of the right leg; he could merely move the toes a little. Sensibility of skin was distinctly lessened on the paralysed side. He had no control over the sphincters. No evidence of disease could be found in other organs.

A few days afterwards he had become able to speak, but did not say more than a few words at a time, being stopped by getting confused. He frequently hesitated and stopped, forgetting the word he wanted to use. He could not name his occupation, but when it was mentioned recognised it at once, and could then repeat the name. When he tried to speak quickly he became confused, mumbled, and became unintelligible.

By the beginning of October his speech had considerably improved and he could move the right leg at the hip and knee-joints. He could not move the right foot or the right hand.

On October 15th he had a succession of fits lasting throughout the day. He was unconscious throughout. His expression was that of pain. He constantly moved his head from side to side. In the fit his right arm was motionless, but at times the muscles were all rigid. The left arm was said by the nurse to be "thrown about" in the fits. Both legs were said to be moved up and down, the left leg more than the right. At times all four limbs became rigid. He was unable to swallow, but remained unconscious till the next day, when, in answer to questions, he said "Yes" and "No."

The attacks continued, becoming less frequent until the 18th, when he had a series of spasmodic twitchings in the right arm and leg, lasting for five minutes, and succeeded by some rigidity.

The rigidity of the limbs continued. It was noticed that the left leg was slightly rigid as well as the right. The right foot was inverted. On October 27th he had so far recovered as to be able to express his ideas well, and answered questions quite rationally.

On October 31st he had become less conscious and an ophthalmoscopic examination was made during sleep without waking him.

Well-marked optic neuritis existed in each eye. No trace of the optic disc could be seen, its position being indicated by a prominent swelling, red in colour, mottled with numerous white dots and patches. The veins were large and tortuous, but could be traced over the swelling to the point of their emergence in the centre. The arteries were small. A few hæmorrhages could be seen outside the swelling.

On November 4th he had another fit, affecting the right arm and leg only. The left arm and leg were motionless, as if paralysed. Tickling the sole caused no movement of leg.

He gradually sank and died from apnœa.

At the *post-mortem* examination a tumour was found in the left hemisphere of the brain. It occupied the greater part of the superior parietal lobule. As a distinct tumour it appeared on the surface only in the lower part of this lobule, where it formed a rounded mass about one inch in each diameter, projecting slightly above the level of the adjacent convolutions. To this portion the pia mater was inseparably adherent. The convolution above this was enlarged, firmer than natural, and on its surface the ramifications of the vessels of the pia mater were deeply impressed. Between the anterior and posterior parts of this convolution an area of apparently healthy substance intervened. A section showed that the growth extended beneath the whole of this area, occupying the white substance and passing up into the convolutions. It extended within a quarter of an inch of the roof of the lateral ventricle, and inwards to half an inch from the inner surface of the hemisphere. The upper extremity of the ascending parietal convolution was not involved, but was pressed upon and narrowed by the growth. Behind the tumour a strip of healthy convolution existed in front of the parieto-occipital fissure.

The outer portions of the tumour were translucent, greyish in tint, dotted with vessels. It passed gradually into the adjacent brain tissue. There was no capsule, no adjacent softening. Where the growth could not be visibly traced the tissue could be felt to be indurated. The central portions of the tumour were greenish-yellow and opaque. This degenerated portion was irregular in shape, somewhat trabecular in aspect, and within it were scattered areas of translucent tissue.

A scraping of the tumour showed under the microscope numerous products of the degeneration of nerve tissue and many small round cells or nuclei $\frac{1}{300}$ inch in diameter, all very granular. On careful

inspection many of these nuclei were seen to be surrounded with a cell-body scarcely larger than themselves, clear and delicate.

On examination of sections of hardened portions the tumour appeared to be made up of minute angular and fusiform cells, $\frac{1}{2500}$ to $\frac{1}{3500}$ inch in diameter, all with delicate dividing processes, which could be traced into, and appeared in great part to compose, the intervening basis substance. These cells were grouped more or less concentrically around the vessels, irregularly elsewhere.

The kidneys were in the early stage of fibroid degeneration. The other organs of the body were perfectly healthy, with the exception of the lungs, which were emphysematous and loaded with blood and serosity. In places the congestion was very intense, and some blood had escaped into the lung substance.

Remarks.—The character of the convulsions in this case is of considerable interest. In many fits the paralysed side was rigid, while the non-paralysed side was the seat of movements which struck the observers as undoubtedly convulsive, and more intense than those of the paralysed side, although they did not consist of simple clonic spasm. In the right hemisphere no lesion was discoverable. The affection of the left side may, perhaps, be connected with the position of the tumour, which was in the midst of and must have involved a large number of the commissural fibres of the corpus callosum passing to the right hemisphere. In a case published last year ('Brit. Med. Journal,' 1874, vol. ii, p. 339, Case III) a small tumour in this situation had given rise to convulsions, the aura of which was at first felt in the opposite wrist and afterwards in the wrist on the same side as that on which the tumour was situated.

The hemiplegia which the growth produced is probably to be explained by its size. Small growths may exist in this situation and give rise to no symptoms of paralysis. The tumour was, however, so large as to extend from the convolutions above to the roof of the lateral ventricle below, and isolated the convolutions on the inner aspect of the parietal lobe.

The limbs on the left side of the body were motionless and apparently paralysed for the day or two preceding his death. No lesion could be found in the right hemisphere to account for this paralysis of the left side, there was no evidence of pressure on the opposite hemisphere, nor was there any considerable effusion

into the lateral ventricles. It may, perhaps, be attributed, like the left-sided convulsion, to an influence of the tumour on the right hemisphere, by means of the fibres of the corpus callosum, the influence being in this instance inhibitory, in the other excitant.

The suddenness of the onset of the paralysis was a remarkable feature in the case. Instead of being, as is usual in tumour, gradual, the early slight and the later complete hemiplegia were sudden in their commencement; the first was attended by slight, the second with very marked loss of consciousness. There was no indication that a convulsion had occurred. No secondary lesion could be found after death to which the suddenness of onset of the paralysis could be attributed.

November 16*th*, 1875.

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8. *Insular sclerosis of brain and spinal cord.*

By James F. Goodhart, M.D.

Harriet B—, æt. 38, was admitted to Guy's Hospital, under Dr. Moxon's care, for the first time in February, 1874, and for the second in December of the same year.

One of her sisters used to stammer when a child, and she also did the same, but got better as she got older.

Ten years ago she had rheumatic fever, and was under treatment in St. George's Hospital. Since then she has remained in good health till two years ago, when she first noticed weakness in her knees. Her attention was first directed to this by finding one day that she was unable to wipe her shoes on the door-mat. Then she found that she had to pay attention to walking, keeping her eyes on the ground to prevent her dragging her legs or stumbling. In this condition she remained until September, 1873, when her hands and arms became affected with shakings, which were only controlled by her clutching anything tightly. She had no pain anywhere, and no feeling of illness. For two months, before her admission, she occasionally had complete loss of control over the legs, owing to stiffness which would come on in them.

No history of syphilis. Catamenia regular. The functions of the bladder and rectum were performed efficiently.

When admitted she had the usual syllabic mode of speech to which such patients are accustomed, and the nystagmus and tremor of the limbs upon any exertion. There was also much rigidity and flexion of the lower limbs, with incapacity of motion or extension. The muscles were not wasted, and electro-motility and sensibility were unimpaired. The irregular jerkings rather increased in severity, and, if anything, became more general, but she had altered but little when she was suddenly taken with shortness of breath, paralysis of the palate, and died.

Sectio cadaveris, forty hours after death.

The skull and membranes of the brain were normal in all respects. The brain weighed 44½ oz. The convolutions were of a nearly normal aspect; perhaps the sulci were rather pronounced, and the membranes stripped very easily. The whole brain felt very hard, and this was especially evident when an attempt was made to separate the two hemispheres and expose the corpus callosum. At the base, too, the structures felt hard, and the medulla oblongata stood out like a rigid column of some inflexible material. Slicing off the hemispheres just above the level of the roof of the lateral ventricles, the characteristic appearances of insular sclerosis at once presented themselves. On both sides the white matter of the cerebrum had patches, mostly circular in form or with circular outline rendered irregular by reason of the confluence of two or more patches of a dark pinkish-grey gelatinous-looking substance. They varied in size, the larger being equal to that of a silver penny. The smaller, very numerous, were no more than a mere point. They were very slightly depressed below the surface of the section, and resisted the knife considerably, retreating like elastic yet tough gristle. Central vessels were not visible. The surrounding white matter was healthy looking.

The parts extensively affected were the roof of each lateral ventricle, the fourth ventricle, pons, medulla oblongata, and upper part of the cord; but the corpora striata and optic thalami, the lower parts of the cord, and even the outlying parts of the brain, had by no means escaped. The cerebellum appeared to have escaped. The grey matter, as a rule, was healthy, though in one or two parts a diseased patch of white matter had run into the grey; but it was possible even then to distinguish the tint of the one from that of the other. The diseased tissue was distinctly darker and more translucent.

The microscopical characteristics of the grey patches in the fresh state were—

(α) Disintegration of the nerve-tubules, which were dark, granular, and fatty looking, huddled up in bundles together. This was the chief change.

(β) Curious shaped bodies, more like epithelial scales than anything else, were found, not numerous in any one spot, but seen in all directions. Some stained deeply with iodine, others not at all. The latter were, it appeared, formed from nerve-fibres rolled up and otherwise changed; those not so were what would be called amyloid corpuscles. Still smaller cells were seen of an inflammatory nature, and they were entangled in a faintly fibrillated substance.

The lungs were somewhat extensively affected with broncho-pneumonia.

The other viscera were healthy.

The case was taken to the Society as a recent specimen.

It is the first recorded in the 'Transactions,' and only, I believe, the third inspection of such a case which has been published in English literature. In German and French works they are more numerous. The other two are published in the 'Guy's Hospital Reports' for 1875, and all three have occurred in the practice of Dr. Moxon. *November 16th*, 1875.

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9. *Myo-lipoma of spinal cord.*

By W. R. Gowers, M.D.

The specimen shown is a small fatty tumour attached to the conus medullaris of the spinal cord, and apparently springing from the pia mater. The cord is that of a patient who suffered from locomotor ataxy, and presents sclerosis of the posterior columns. The specimen is interesting from the extreme rarity of a fatty tumour in this position and from the circumstance that it contains striated muscular fibres.

The tumour measures half an inch from above down, half an inch from before back, and about three eighths of an inch from side to side at its thickest part. Its transverse section is somewhat cre-

scentic in shape, its concave side embracing one half of the cord and extending from the anterior to the posterior fissure (*vide* Pl. I, fig. 1). Many of the nerve-roots of the cauda equina are embedded in it. When fresh the tumour was of a yellowish colour and in consistence and aspect resembled a fatty tumour. Such it proved on microscopical examination (*vide* Pl. I, fig. 2, and Pl. II, fig. 1). It is composed entirely of round and oval fat-cells closely pressed together, and precisely resembling those of ordinary adipose tissue. They measure $\frac{1}{300}$ to $\frac{1}{500}$ of an inch in length, and contain no crystals. They are for the most part in contact one with another, but at the angles the interstices are occupied by a delicate finely granular and fibrillar tissue, in which here and there oval nuclei $\frac{1}{4000}$ inch in diameter are to be seen. No nuclei are visible in the cells themselves. In places the cells are separated by tracts of fibrous tissue, the fibres being wavy, fine, and dotted here and there with nuclei. These are very abundant on the inner portion of the tumour adjacent to the spinal cord. Many of them are continuous with the fibrous pia-matral layer which separates the lateral columns of the cord from the substance of the tumour. This is a little thickened, but not otherwise different from that of the opposite side. From its outer surface these tracts of fibres pass into the substance of the tumour, some to unite again, enclosing spaces filled with fat-cells, others to ramify among the adjacent fat-cells.

The tumour is enclosed in a capsule about $\frac{1}{200}$ of an inch in thickness, composed of fibrous tissue identical in appearance with that of the pia mater. This capsule increases in thickness on the front and back of the tumour towards the inner side, where it blends with the connective tissue in the openings of the anterior and posterior fissures, and even extends for a short distance on to the opposite anterior and posterior columns of the cord. The fat-cells of the tumour are smaller and are less closely pressed together near the capsule than in the centre and inner part of the tumour.

Many of the nerve-roots of the cauda equina on the left side are seen embedded in the tumour. A few lie on the surface, but more are enclosed in the mass. They lie chiefly in its anterior and posterior portions.

The fibres of the posterior roots present marked degeneration. Those of the anterior roots appear perfectly healthy.

In the anterior part of the tumour is much wavy fibrous tissue, running for the most part transversely.

DESCRIPTION OF PLATE I.

Plate I illustrates Dr. Gowers's specimen of Myo-lipoma of the Spinal Cord. (Page 19.) From drawings by himself. (See also Plate II, fig. 1.)

FIG. 1.—Section of tumour and conus medullaris. × 8.

a a. Groups of muscular fibres.
b b. Bundles of nerve-fibres of cauda equina.
c. Muscular fibres, running transversely, mingled with fibrous tissue. (These are shown more highly magnified in fig. 2.)

2.—A small portion of the tumour and of lateral column of cord. × 60.

d. Portion of lateral column of cord.
e. Thickened pia matral tissue, sending fibrous processes into tumour.
f f. Tracts of wavy fibrous tissue.
g. Fat-cells of tumour.

Fig 1

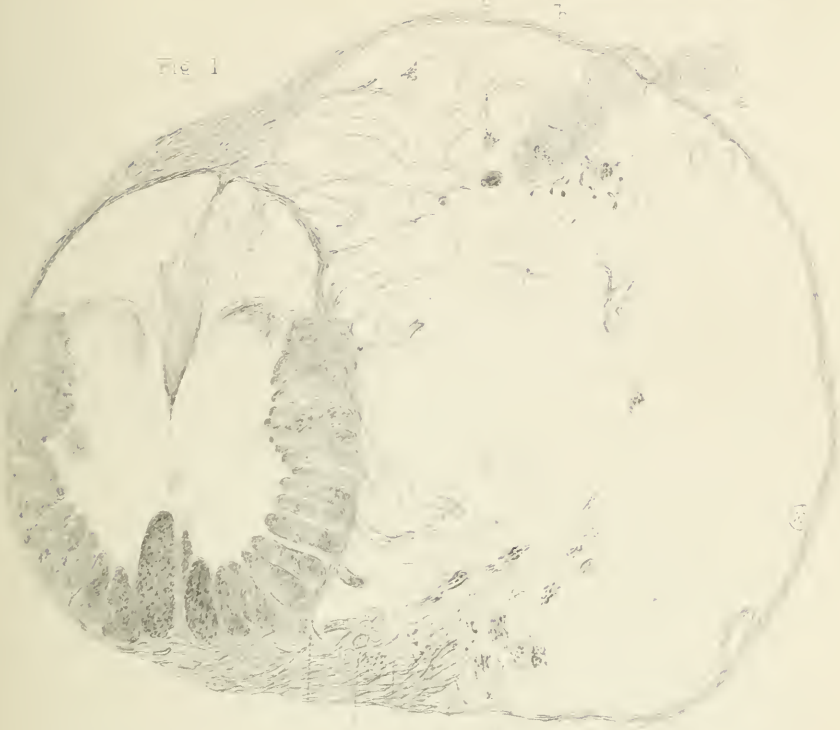


Fig 2

In addition to these elements within the tumour are many striated muscular fibres (*vide* Pl. II, fig. 1). They occur in groups or isolated. They are most abundant in the anterior part of the tumour, among the fibrous tissue which is so abundant there, and occur in close contiguity to the anterior nerve-roots. Other groups of these fibres are situated near the posterior nerve-roots and a few are scattered in the intervening fatty tissue. Their direction is for the most part vertical, but in the fibrous tissue of the anterior part of the tumour many fibres run transversely. In diameter these fibres vary from the $\frac{1}{900}$ to the $\frac{1}{2500}$ of an inch. The transverse striation is perfectly characteristic, being identical with that of the fibres of voluntary muscle. The striæ are for the most part perfectly regular, but here and there are a little irregular. Their distance apart varies in different fibres; the average distance is the $\frac{1}{8000}$ or $\frac{1}{9000}$ of an inch, but on some of the smaller fibres they are $\frac{1}{6000}$ or $\frac{1}{7000}$ inch apart. In some fibres, chiefly of larger size, their distance apart is not more than $\frac{1}{10000}$ inch. Here and there fibres are seen resembling the striated fibres, but presenting only an irregular longitudinal striation; these, it is probable from their identity in size and course with other adjacent striated fibres, are empty fibre sheaths.

A few large vessels traverse the tumour in various directions.

No other abnormality of the membranes of the cord could be found.

Remarks.—The structure of the growth thus resembles that of fatty tumours as found elsewhere, with the addition of rather an unusual quantity of fibrous tissue and with the very rare addition of striated muscular fibres. Its fibrous capsule and framework are continuous with the tissue of the pia mater, and it appears to have originated in the cellular bands and membranes which connect the outer surface of the pia mater with the arachnoid.

As far as I have been able to ascertain, no other instance of lipoma within the dura-matral sheath of the spinal cord has ever been recorded. Very few examples have been met with of fatty tumours connected with the nerve-centres, and those have been connected with the cerebral dura mater, the cerebral pia mater, or the ependyma of the lateral ventricles. Most of them are referred to in Sir James Paget's 'Surgical Pathology;' none were connected with the spinal cord. This rarity of such specimens may be associated with the invariable freedom of the tissues within the dura matral sheath from fatty infiltration. Outside the spinal dura mater there is abundance of

adipose tissue; within it none. The brain and spinal cord share this peculiarity with some other viscera, *e.g.* the liver. But the fact seems worthy of remark when it is considered how large a share fatty material takes in the composition of the nervous tissue. Moreover, recent researches (of Axel Key and Retzius) have demonstrated the cellular structure of the subarachnoidal tissue, and it might be conceived to offer a ready field for fatty infiltration. But this seems never to occur.

Can the fatty tumour and the degeneration of the posterior columns of the cord be regarded as standing in any causal relationship? Probably not. The tumour could not by its pressure have caused the degeneration of the posterior columns, because (1) the growth was situated at the side, not at the back of the cord. (2) The degeneration involved the whole width of the posterior column, not only in the lumbar region but also throughout the cord; had it been an ascending degeneration in the upper part of the cord it would have been confined to the posterior median columns. (3) It may be doubted whether simple fatty tumours ever cause damaging pressure upon *organs*. The effect of pressure is to limit the infiltration of the cells of the growth, rather than to injure a resisting structure. On the other hand, the extreme rarity of fatty growths on the subarachnoid tissue and the commonness of degeneration of the cord would render it unlikely that the tumour originated from the fat liberated by the degeneration of the nervous elements. The two morbid conditions were probably accidentally coincident.

Moreover, the presence of striated muscular fibres in the tumour suggests strongly the probability of its congenital origin. These fibres occur with great rarity in morbid growths, and I am not aware that their occurrence in a fatty tumour has been before observed.

December 20th, 1875.

10. *Tumour of the sciatic nerve.*

By Sidney Coupland, M.D., for Daniel Balding.

George E—, æt. 34, was admitted into the Royston Cottage Hospital on June 24th, 1874. "For the past three years he has complained of pain in the left leg as high as the knee, and for twelve months preceding his admission he has noticed a swelling at the back of the limb, about three inches above the knee, in the median line. The swelling had increased much in size during the last few months, and now (*i. e.* at the time of admission) it causes him so much pain as to prevent him from following his occupation as a farm labourer. It is hard, fairly movable, not very tender, and apparently not adherent either to the skin or to the bone beneath. No fluctuation can be felt and no bruit heard in the tumour. The patient's general health is good."

July 21st, 1874.—Chloroform being administered, an incision was made in the upper part of the ham, over the tumour, which was rapidly displayed. It was seen to be spindle-shaped, and to measure about four to five inches in length and one inch and a half in width, its long diameter being parallel to the axis of the limb. It appeared to be ensheathed within the great sciatic nerve, the fibres of which could be seen passing over it. The tumour being dissected out, its removal was effected by dividing the nerve above and below the growth. The whole length of nerve thus removed measured about five inches. There was free bleeding from a vessel of considerable size seated in the nerve at the point of section above.

The operation was followed by a considerable amount of cellulitis in the thigh, as high as the greater trochanter, accompanied by extensive suppuration.

The condition of the limb at the end of six months was briefly as follows:—The knee is semi-flexed, but movement of the joint is preserved. There is no movement at the ankle-joint, the foot is inverted and toes extended. There is much muscular wasting of the leg, and walking about much with crutches produces considerable œdema of the leg and lower third of the thigh. Sensibility is retained on the front of the leg and dorsum of the foot, but is abolished on the back and sole. A slough which formed on the left heel has now healed up. At the upper part of the cicatrix in the thigh there is a mass of induration over the site of the extremity of

the divided sciatic nerve. At this point there is some tenderness. The patient refuses further operative measures.

January 4th, 1876.

Report by the Committee on Morbid Growths upon Mr. Balding's specimen of tumour of the sciatic nerve.—The specimen presented to us for examination consisted of a bisected tumour of ovoid form, about three inches long by one and a half inches wide, enclosed in an ill-defined fibrous capsule, continuous at each end with the sheath of the sciatic nerve, a small portion of which existed at the upper and lower ends of the tumour. Some few fibres of the nerve were spread over the surface of the tumour, but the greater portion were collected into one large bundle. The cut surface of the tumour showed one large mass, comprising nearly the whole of the growth, and several very small portions at one end more or less completely separated from the first. Seen with the naked eye the surface was variously mapped out by two or three large, irregular, longitudinal bands, giving off numerous smaller transverse bands. Other general characters of the tumour were not well seen, owing to its having been some time in spirit.

Microscopical examination of sections in dammar and glycerine showed trabeculæ of fibrous and spindle-cell-tissue, for the most part having a circular arrangement, but occasionally forming thick longitudinal bands (*vide* Pl. II, figs. 2, 3, 4, 5). The circularly disposed trabeculæ enclosed small areas containing mucous tissue, with a faintly granular basis and very numerous cells, arranged concentrically within each area. The cells presented every variety of form, from round and oval well-formed cells to others branched in the most diverse manner, the processes of the latter anastomosing freely. The basis-substance contracting and separating from some of the round cells gave the appearance of a double contour, resembling the characters of cartilage-cells. The size of the cells varied as much as their form, the round cells being generally small, whilst many of the branched cells were of considerable size. The small cells were chiefly found in the smaller areas, the large cells lay in great abundance in the larger areas. The nucleus of each cell was single and often not very distinct.

From its microscopical characters we should be inclined to consider this tumour a fibro-myxoma.

Henry Trentham Butlin.
Rickman J. Godlee.

DESCRIPTION OF PLATE II.

Fig. 1 illustrates Dr. Gowers's specimen of Myo-lipoma of the Spinal Cord. (Page 19.) From a drawing by himself. (See also Plate I.)

The drawing shows striated muscular fibres and fibrous tissue from the anterior part of the tumour. × 250.

Figs. 2, 3, 4, and 5 illustrate the Report by the Committee on Morbid Growths on Mr. Balding's Tumour of the Sciatic Nerve. (Page 24.) From drawings by Mr. Butlin.

Figs. 2, 3, and 4. Hartnack, oc. 3, object. 7.
5. Hartnack, oc. 3, object. 4.

Fig. 1

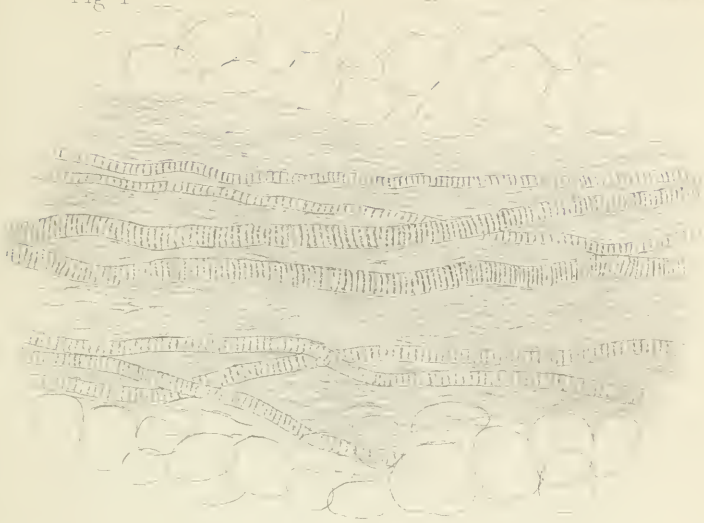


Fig. 4

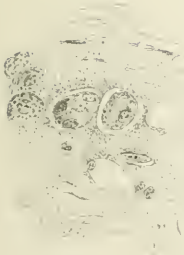


Fig. 3

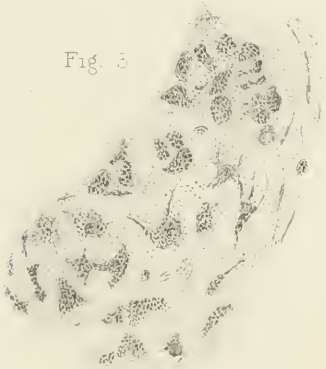


Fig. 2

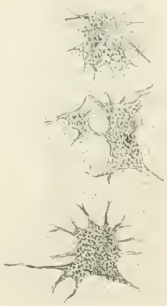
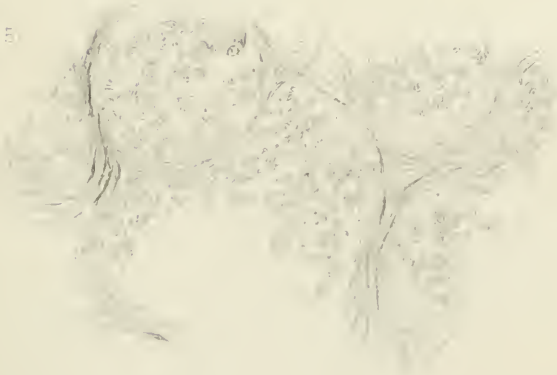


Fig. 5



11. *Cyst of the choroid plexus of large size in an infant.*

By W. Cayley, M.D., for George Brown.

The pathological specimen which I have, through the kindness of Dr. Cayley, the honour of submitting to your notice this evening, was removed from the brain of an infant, four months old, who died after an illness of about ten days, the chief symptom of which was persistent vomiting.

The cyst, which sprang from the choroid plexus of the left ventricle, distended and completely filled that cavity, and had forced its way through the corpus callosum and between the hemispheres, protruding beyond the surface of the cerebrum so as to touch the dura mater, to which membrane it had formed a slight adhesion. When removed from the brain it was as large as a full-sized duck's egg. Its walls were extremely thin and translucent, and it was full of clear serous fluid, which, however, rapidly escaped from the interior of the sac by exosmosis. The choroid plexus on the right side was quite healthy.

As the case is of some clinical interest I may, perhaps, be permitted to add a few notes to the above brief description of the tumour.

The patient, Archibald J—, had been under my care since he was two months old for severe eczema of the head and face, otherwise he was to all appearance a healthy, fully developed, and well-nourished child. The only thing about him to excite suspicion of internal disease was the fact that he frequently cried in a peculiar whining manner, as if in pain.

With respect to family history, he was the youngest of a family of six children, two of whom died in infancy; one, the first-born, of atrophy, at three weeks, and the other, the fifth child, of acute tubercular meningitis when one year and eight months old. One of the children now alive is rickety, the others are healthy. Both father and mother are healthy.

On October 15th the patient was brought to me on account of persistent sickness. The mother stated that he had been unable to keep anything on the stomach for three days. I saw the child on the next day; the bowels had acted freely, but the vomiting continued unabated. Ordered a sedative mixture.

Again saw the child on the 18th. The vomiting still continued.

The abdomen was distended with flatus, and pressure on any part of it appeared to cause much pain. There were no convulsions or paralysis, and beyond the persistent vomiting there was no sign or symptom to lead me to suspect cerebral lesion. Ordered warm poultices over the abdomen, a sedative mixture, and Brand's essence of beef to be given instead of milk.

I did not see the child again for two days. Meantime the idea occurred to me that the vomiting might perhaps be due to cerebral mischief, and it appeared to be the more probable that such might be the case as the mother had lost a child a few months previously from what the medical attendant believed was acute tubercular meningitis.

On October 20th, the ninth day of the illness, I found a great alteration in the child. He was very much wasted, and had an aged appearance. He took no notice of any person or thing; the pupils were widely dilated, and there was lachrymation of the left eye; no paralysis or convulsions. The tympanitis had entirely disappeared, and the abdomen was much retracted. Since my last visit he had taken two quarter-pound tins of Brand's essence of beef by teaspoonfuls without being sick afterwards. The sickness returned, however, if milk were given. He still continued to whine a good deal, and had an expression of countenance indicating pain. There was no bulging at the fontanelles, but the left parietal eminence was markedly more prominent than the right. At the time I attached no importance to this sign, as I believe it is not at all uncommon in children to find unsymmetrical development of the bones of the skull. At the *post-mortem* examination I found that the situation of the tumour corresponded exactly with this prominence.

The child never rallied, but gradually sank, and died on the next day (October 21st) without any convulsions or struggling.

It appears somewhat extraordinary that such a large cerebral tumour could have existed, as it must necessarily have done, for a considerable time, perhaps from birth, without giving rise to more marked symptoms. This may, perhaps, be explained by the fact that the bones forming the vault of the skull were in this case so soft that, as the tumour enlarged, the roof yielded before it, and thus there was scarcely any pressure on the base of the brain. Had such a tumour occurred in a child whose cranial bones had become ossified, there is no doubt that well-marked symptoms would have been manifested. *January 18th*, 1876.

12. *Chronic hydrocephalus in an adult, apparently the result of fracture of the skull.*

By P. H. Pye-Smith, M.D.

The man to whom the brain exhibited belonged came under my care last November, having been previously a patient of Dr. Wilks. He was the subject of regurgitant and obstructive disease of the aortic valves from rheumatism, as well as of pleurisy and bronchitis, aggravated by deformity of the thorax, and to these affections was due the fatal result of his illness.

The only symptom observed which appeared to be of cerebral origin was slowness of the pulse, which, in spite of the advanced cardiac disease, was never above 68, and often as low as 40. There was no paralysis, vomiting, headache, or mental disturbance. He had, however, been previously subject to epileptic attacks. The first, of which the account was imperfect and the character doubtful, happened five years before his death, and three years after a fall into a sawpit, when he cut his head and was "insensible and delirious for two days," but recovered with no other effect than deficient memory. Three years before his death, *i. e.* when he was thirty-two years old, a brick fell on his head and again cut it open. He then attended as an out-patient at St. Bartholomew's Hospital, and did not suffer from cerebral symptoms.

For eight weeks before he was admitted into Guy's Hospital he had suffered from repeated attacks of *petit mal*, marked by pallor, unconsciousness, and twitching of the limbs, lasting about five minutes, and followed by headache. He was at last attacked by a series of much more severe epileptic fits, with foaming at the mouth, biting the tongue, lividity, and convulsions, which after several hours left him in a semi-comatose state.

It was when recovering from this condition that he came under Dr. Wilks's care, and, as above stated, had no fits or other cerebral symptoms during the four months he lived in the hospital.

After death, beside the cardiac and pulmonary lesions recognised during life, the ventricles of the brain were found distended as here shown. The convolutions were, of course, flattened, but the cortex appeared everywhere normal, except for a few minute rust-coloured

spots, chiefly on the under surface of the frontal lobes. The pia mater covering the temporo-sphenoidal lobe was also stained of a reddish-brown colour. The ependyma was thickened, but not granular, and the fornix and septum lucidum were of normal consistence. There were no signs of chronic or acute meningitis. The fluid in the ventricles weighed fourteen ounces. It was clear and free from albumen, fibrin, or blood. The brain itself weighed forty-eight ounces. The aqueduct of Sylvius was patent, and there was no evidence of obstruction about the veins of Galen.

The skull showed a fracture which will be seen to run partly in the line of the more or less obliterated right lambdoidal suture, and then outwards and downwards for half an inch between the mastoid and squamosal parts of the temporal bone. The foramen magnum was of unusual shape, the transverse diameter being greater than the antero-posterior.

The rarity of hydrocephalus in an adult has led me to put this case upon record. The fluid was cerebro-spinal, so that it was a passive not an inflammatory effusion—a true dropsy of the brain. That the fracture of the skull received either eight or three years before death was in some way the cause of increased venous pressure appears probable, though we could find no trace of adhesions or other obstruction to the return of blood from the choroid plexus.

April 18*th*, 1876.

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13. *Meningitis.*

By Edwards Crisp, M.D.

Tuesday, March 15th, 1876, I was called at 9½ p.m. to see Miss S—, æt. 11, whom I have known for some years. She is a very tall child for her age; was delicate in infancy, but has since enjoyed a fair share of health. She has never been subject to headache. Her mother gave the following history:—"On the day before (Monday) about two o'clock she had returned from school in excellent spirits, and told her mother in a laughing way that she had had a fall, and that she fell *flat*, but did not complain of any pain or

hurt from the fall; she was alone at the time, but probably she fell upon the pavement. After this she ate a good dinner. In the evening she was a little sick, and complained of pain in her head, referring it more to the left side over the ear. On the following morning, Wednesday, she still complained of pain in the head, with sickness. When I was called at night she had great pain in the left side of the head, extending over the vertex; the skin was hot, the pulse quick and jerky. The mother said she had had slight discharge from the left ear for a day or two, but on examination of the ear I could find no indication of this.

Thursday, 16th.—Has passed a very restless night; the pain in the head now extends across the forehead and vertex to the right side; screamed frequently during the night, and at times was delirious. Skin hot; tongue coated and dry; temp. 103° F.; pulse quick, but less jerky than last evening; great thirst; wild expression of countenance and staring eyes; pupils rather large, but equal in size. There is great heat of the scalp, and she complains of pain along the upper part of the spine; extreme restlessness; no arching of the body, but slight subsultus of the fingers. There is no unusual tenderness over the spinal column when pressed upon, except in the region of the cervical vertebræ, and here the pain is not great.

I saw her again at twelve; told her parents that she was in great danger, and requested the opinion of Dr. Murchison, who was unable to see her until ten the same night. Dr. Murchison, like myself, thought her in great danger, but there was no appearance of immediate death. When we (Dr. Murchison and myself) were leaving the house the mother called us back, as she observed a sudden change in the child; the pulse became imperceptible, and she died in about a quarter of an hour (10.30), about twenty-five hours from the time when I first saw her.

Post-mortem examination.—Skin of a normal appearance; no eruption and no discoloration in any part. The dura mater normal; arachnoid dry and rather opaque, with numerous yellowish-white patches (as seen in the wax cast) between it and the pia mater. This appearance extended over the cerebrum and cerebellum, and was not more seen on the left side than on the right. The vessels on the surface of the brain were much distended. The brain was very carefully sliced, but no abnormal condition, irrespective of that described, could be detected. There was a very small quantity of serum in the lateral ventricles. The petrous portion of the temporal bone on both

sides was sawn through and carefully examined, but no appearance of inflammation or of purulent matter could be detected.

The brain substance, as far as I could judge, was not perceptibly altered. The cerebellum weighed $5\frac{1}{2}$ oz., a great weight for a child of this age.

We were not permitted to examine any other part of the body; but, as far as I could judge from the symptoms, the other viscera were in a healthy condition, there being no indication of lesion in any other organ; indeed, the symptoms, as supposed by Dr. Murchison and myself, were all referable to membranous inflammation of the brain.

Remarks.—Opinions may vary as to the exact nature of this case, but, as I have said above, to me it was evident the brain membranes were the chief seat of the lesion. If the spinal cord had been implicated, probably convulsive movements of the limbs would have been present. I have seen only four cases of cerebro-spinal meningitis, but judging from these, and more especially from the perusal of a great many cases, I am not inclined to place it under this category. The ear discharge, which at first led me to think of inflammatory extension to the cerebellum, was shown by the autopsy to have little or nothing to do with the meningitis. I am disposed to think that the fall, which might have been more severe than was at first supposed, was the cause of it. The rapid sinking was, I think, a remarkable feature in the case, as the heart's sounds afforded no indication of mischief in that organ. *April 18th*, 1876.

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14. *Posterior sclerosis and posterior median sclerosis of spinal cord.*

By W. R. Gowers, M.D.

Posterior sclerosis.—The sections are from the spinal cord of a man, æt. 38, who suffered from locomotor ataxy, and died in University College Hospital, under the care of Sir William Jenner, in 1869. There was no neurotic family history, no history of sexual excess. The first symptoms of unsteadiness dated from three years before his death, when he found himself liable to fall forwards when

he closed his eyes in washing. A year before death it was noted that the muscular power of his legs was good, that he was able to resist flexion and extension strongly, but that his walk was extremely unsteady, the unsteadiness being increased by closure of the eyes. There was considerable inco-ordination of movement of the hands, so that he could not pick up a pin. The only defective sensibility noted was in the soles of the feet, where he could not define the spot pricked with a pin. There seemed some impairment of sensibility to heat, especially in the right limbs, but the results were not very conclusive. He complained of a feeling of "numbness" in his fingers and toes, rather greater in the right hand than in the left. Urine retained. He was not seen again for twelve months, and then was dying from the results of an intercurrent chest affection (empyema).

The spinal cord presents, through its whole length, uniform fibroid degeneration of the posterior columns. The anterior and lateral columns are free from change; the anterior cornua are normal, and contain large, healthy-looking nerve-cells. Here and there are large spaces around the vessels in the grey matter. At the level of the eighth cervical nerves, there is some disintegration of the right cervix cornu posterioris. In the cervical region the posterior median columns (cuneiform columns) contain the largest tracts of fibrous tissue. The front part of the posterior columns are almost free from sclerosis in their outer part, adjacent to the cervix cornu posterioris. At the highest part of the cervical region, where the caput cornu posterioris begins to enlarge, the sclerosis is much less marked in the anterior and posterior parts of the posterior columns, being chiefly confined to a band which stretches across them. In the dorsal region the fibroid change is uniform and dense throughout the posterior columns. In the upper lumbar region the sclerosis of the posterior column is coarsely fasciculated in the anterior part, and fine and uniform in the posterior portion. The posterior nerve-roots seem here largely implicated in the degeneration. Towards the middle part of this region no trace of nerve-fibres can be seen in the hinder half of the posterior column. The degeneration extends to the lowest part of the conus medullaris. To the upper part of the conus was attached the peculiar myolipoma described at a previous meeting (see p. 19).

Posterior median sclerosis.—The second set of sections exhibit a sclerosis of the posterior columns which in the lumbar region extends

through the whole width of the columns, but in the dorsal and cervical regions is limited to the posterior median columns.

The cord is that of a young man, æt. 30, who died in a series of epileptiform convulsions, beginning unilaterally in the left hand, he having had, three months before, a similar series of fits. He is not known to have presented any cord symptoms. Certainly, when under observation three months before his death, any ataxic symptoms must have been such as to escape notice, and during the succeeding three months he was believed to be in good health. The cord was examined to ascertain if any lesions were left by the violent convulsive seizures, and the discovery of the sclerosis was thus accidental.

In the lower lumbar region the whole width of the posterior columns is densely sclerosed, comparatively few nerve-fibres being seen. In the upper lumbar region the sclerosis is dense in the anterior portions of the posterior columns, and dense behind in the median portion of those columns, but becomes much less close in the external tracts adjacent to the posterior cornua. Throughout the dorsal region the external portions of these columns are almost healthy. The change is there confined to the posterior median columns (cuneiform columns), a narrow tract on each side of the posterior median fissure, widening out behind, its outer borders being nearly parallel to the inner boundary of the posterior cornua. In this region scarcely any trace of nerve-fibres can be seen. At the level of the eighth cervical nerves the change is confined to the same columns,but a little higher up there is a considerable excess of fibrous tissue in the anterior portion of the posterior columns throughout their entire width, which has an appearance as if it were spreading from the anterior part of the sclerosed cuneiform column. The change exists up to the highest part of the cervical region.

The anterior and lateral columns throughout the cord are free from anything more than a uniform slight excess of connective tissue.

The anterior and posterior cornua also appear healthy.

No visible lesion was found in the brain. The other organs (including the kidneys) were healthy.

Remarks.—The changes in the first of these cords is that constantly found in ataxy affecting both upper and lower limbs—fibroid degeneration of the posterior columns of the cord in their whole extent. The second cord illustrates the fact that the sclerosis of

the posterior median column does not cause inco-ordination. This fact has been clearly established by the labours of French pathologists, especially MM. Vulpian and Pierret, &c., but I do not think an example of it has hitherto been brought before this Society. The degeneration of these columns in the cervical and dorsal regions is simply an ascending degeneration due to the more extensive destruction of the posterior columns (from any cause) lower down the cord. It is constantly seen in cases of locomotor ataxy, in which the whole posterior columns are sclerosed in the lumbar region. In this case there seems sufficient change there to account for the ascending degeneration. So marked, indeed, is this that it is impossible to believe that ataxic symptoms can have been altogether absent. Instances are frequent in which the early symptoms of ataxy are overlooked.

In the cervical region in one part the appearance, just described, is as if the fibroid change were spreading from the anterior part of the postero-median tract into the adjacent anterior part of the postero-external columns. It is possible that such extension may be the way in which the postero-external columns in the cervical region become affected. At any rate, the appearance affords some justification for the statement of Niemeyer, that the sclerosis of ataxy may commence in the postero-median columns, a statement which, in the light of recent study of the ascending degeneration, has been rather severely criticised.

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15. *Cerebral aneurysms, associated with endocarditis.*

By W. R. Gowers, M.D.

This specimen of cerebral aneurysms is of interest on the one hand from their association with endocarditis, and on the other from the rare form of ventricular hæmorrhage to which the rupture of one of them gave rise, and from the paralytic symptoms which a previous cerebral hæmorrhage produced.

The patient was a girl, æt. 17, admitted into the Hospital for the Paralysed and Epileptic on April 7th, with almost complete left hemiplegia. No history could be obtained of rheumatic fever,

scarlet fever, or chorea. Eight months before admission she had been much frightened by seeing a horse run away. She was menstruating at the time, and the menstrual flow ceased suddenly immediately afterwards and never returned. Soon afterwards she began to suffer from pain in the left side of the chest, palpitation of the heart, and slight swelling of the legs. These continued increasing until the hemiplegia.

The onset of the hemiplegia was sudden one month before admission. She went to bed as well as usual, and woke at four in the morning screaming "Oh dear! oh dear!" She says she was alarmed by a "trembling feeling," and that immediately afterwards something seemed to burst in her head, "like a bladder bursting," and some fluid ran out of each ear. (There had been no earache.) Immediately afterwards she became unable to speak, and then the left arm and leg became motionless. There was, apparently, no loss of consciousness. Power of speech was soon recovered, and in about ten days there was slight power of moving the leg.

On admission she was still unable to stand, but could move, a little, all the joints of the leg. The arm had less power; at the shoulder-joint the arm could be moved forwards and backwards, but could not be abducted. The elbow-joint could be flexed and extended; the fingers could be flexed a little, not extended. There was slight rigidity of the limb. The eyes were straight; there was no conjugate deviation. The masseter contracted less strongly on the left side than on the right, but there was no defect in the lateral movements of the jaw.

The face at rest was almost equal. The ocular fissures were quite equal, and so, as a rule, were the angles of the mouth; sometimes the right angle was a little sharper than the left. The movement in the upper part, forehead muscles, and orbiculares, was equal on the two sides. In the lower part there was a remarkable amount of paralysis of voluntary motion. When told to raise her upper lip it was raised well on the right side. The naso-labial furrow was deep, the skin inside of the nose was thrown into wrinkles, and the eye was closed a little. The left side of the face, however, remained almost motionless, the lip was not raised at all, and the naso-labial furrow could not be seen. The skin on the nose was not moved. The difference between the two sides was almost as great as in a case of peripheral facial paralysis. The immobility was scarcely less when she tried to widen the mouth by moving the corners outwards.

Nevertheless, in a spontaneous smile or laugh there was scarcely any difference between the two sides; the right side moved a very little more than the left. In smiling, however, the right orbicularis palpebrarum contracted more readily than the left (markedly so), but in the stronger movement of a laugh the two were equal.

In the mouth the tongue was straight and was pushed against one cheek as readily as against the other. Out of the mouth it protruded considerably to the left.

There was no defect in the field of vision or in the other special senses. The whole of the left side presented a little hyperæsthesia; a touch was felt more readily and a slight pinch occasioned more pain.

The urine contained one-third albumen, with hyaline and granular casts. There was œdema of the left limbs, not of the right. A slight pinch produced an extravasation. Her pulse was small and frequent (120), of moderate tension. The heart's impulse was diffused; the apex beat lowered. There was at the apex a loud, rough, systolic murmur, audible at the base, but less loud; faint at pulmonary cartilage, and still more faint at the aortic cartilage; heard well at the back. Second sound accentuated.

During the next three weeks the patient improved in general state and acquired a little more power over the paralysed limbs. On the morning of the 28th she complained of giddiness, and in the evening she had a fit. It commenced suddenly with spasm in the two hands and arms. When Dr. Sturge saw her a few minutes afterwards he remarked that this was clonic and equal on the two sides. The legs and face were not moved in the convulsions. The face was flushed, the carotids were beating violently, the surface was warm and perspiring. The fit lasted for ten minutes and she then recovered consciousness and was violently sick. The next day she was better, but on the following day (30th) she had two slight fits, and in the evening some severe ones. In the first there was violent general clonic spasm and in the second the face was drawn to the right. The face was flushed and there was much palpitation of the heart. She recovered consciousness completely, but in a few minutes she had another short fit and after this became comatose, and remained comatose until her death, twelve hours later.

Post-mortem examination.—The brain contained two separate foci of hæmorrhage, one recent, the other of some standing, both in the

right hemisphere. The older hæmorrhage was situated in and beneath the upper half of the transverse frontal fissure (præcentral sulcus). On the surface the extravasation was one-third of an inch in greatest width, and had separated and pressed upon, without lacerating, the convolutions which bound this sulcus, *i. e.* the ascending frontal convolution behind and the posterior extremities of the superior and middle frontal convolutions, in front. The clot extended beneath the surface for about an inch into the substance of the hemisphere. It was of a uniform chocolate colour, and presented under the microscope chiefly granules and aggregations of brownish pigment-granules. No source of the hæmorrhage could be discovered. It was quite isolated.

The more recent extravasation consisted of a large mass of black clot, mingled here and there with paler portions, which lay outside the corpus striatum, between it and the convolutions of the operculum (in the angle between the two portions of the fissure of Sylvius). It was about two inches in each diameter, and corresponded in length to the nucleus caudatus. The clot and the caudate nucleus were close together behind, but diverged in front, where they were half an inch apart. More deeply the clot approached close to the outer side of the lenticular nucleus, but did not invade it. The clot reached the surface of the brain, in the fissure of Sylvius, beneath the operculum, where it was in contact with an aneurism presently to be described. Internally the clot was continuous by a narrow band of black coagulum, with a strip of similar coagulum which lay in the right lateral ventricle in the depression between the corpus striatum and optic thalamus. This coagulum was not adherent, was quite smoooth, and extended backwards as far as the commencement of the descending cornu, where it ended in a broad extremity. Anteriorly it also widened out so as to form a film three quarters of an inch wide over half the corpus striatum. Through the foramen of Monro it was continuous with a similar but narrower band of similar clot, which occupied a similar position (in the depression between corpus striatum and optic thalamus) in the opposite lateral ventricle, also ending in an enlarged extremity at the commencement of the descending cornu. At the foramen of Monro a continuity of clot extended into the third ventricle, passing as a narrow band through it into the passage to the fourth ventricle, where it lay over the whole floor and passed by the foramina to the subarachnoid space. From the surface clot in the fissure of Sylvius a small quantity of

coagulum extended along the fissure to the base of the brain, where it lay beneath the arachnoid in the interpeduncular space (not, however, distending it), and formed a thin film over the upper half of the pons. This was not continuous with the clot which had escaped from the fourth ventricle.

The arteries at the base of the brain were healthy, only the slightest possible evidence of commencing degeneration being here and there visible. Within the fissure of Sylvius, lying against and embedded in the clot at the spot at which it appeared at the surface, was an aneurism one third of an inch long, oval, and filled with firm laminated clot. It was attached to the second branch of the Sylvian artery, namely, that which goes to the ascending frontal convolution. The artery beyond was not obliterated, but on one branch, about $\frac{1}{50}$ inch in diameter, the microscope revealed an oval aneurismal dilatation, distended by clot. Another large aneurism, about one quarter of an inch in length, was found lying in the upper part of the fissure of Rolando, attached to a branch or to the trunk of the third branch of the Sylvian artery, viz. the ascending parietal convolution. This aneurism had a somewhat thick wall, and was also filled with clot.

The *heart* was large, weighing $13\frac{1}{2}$ ounces. The right side was healthy. The mitral orifice measured $4\frac{1}{2}$ inches in circumference, and on its auricular surface was a row of large wart-like vegetations. Those on the aortic segment were in the usual position at the edge of the valve, but on the other segment they were situated at the edge of a fold which projected inwards across the middle of the flap. On the posterior surface of the auricle was a patch of similar vegetations, half an inch in diameter. The left ventricle was dilated; the average thickness of its wall was half an inch, the length of its cavity 4 inches, its mid-circumference $5\frac{1}{2}$ inches. The aortic valves were healthy; circumference of orifice, 3 inches.

The kidneys were both very large and pale. The right measured 5 inches by $2\frac{3}{4}$, and weighed $8\frac{1}{2}$ ounces; the left measured 5 inches by $2\frac{1}{2}$, and weighed $7\frac{1}{2}$ ounces. The surface was smooth; the capsule a little adherent; the substance pale and opaque, mottled by vessels; consistence lessened. Under the microscope the tubes were distended with granular epithelium.

The liver was large, and weighed 4 pounds 3 ounces, and was very pale, smooth, and the cells were infiltrated with fat-globules.

The spleen was large, measuring $7\frac{1}{2}$ inches by 4, and was softened. In its substance were several old caseous infarctions.

The lungs were congested.

Remarks.—The general state accords best with the supposition that the patient had a slight unrecognised attack of scarlet fever (then epidemic) about six months previously, which led to nephritis and endocarditis, leaving a large pale kidney and diseased mitral valve.

The first point of special interest in the case is the association of the valvular disease of the heart with the cerebral aneurisms. The frequency of this association has been remarked by several observers, and was first pointed out by Dr. Church, who founded upon it the theory that many of these aneurisms are due to imperfect plugging of vessels by embolism. This case, I think, affords considerable support to that theory. In it the vessels of the brain, as a whole, were healthy; the patient was young, there was no general vascular degeneration. There were certainly three aneurisms, perhaps four, for it is probable that the older hæmorrhage, in so rare a seat, was due to the rupture of a similar aneurism which was no longer discoverable. All these aneurisms were situated on branches of one of the cerebral arteries, and that the artery most frequently obstructed by embolism. There was, further, the evidence afforded by the splenic infarction that embolism had taken place in the system some time before.

There can be no doubt that this aneurism was the source of the large hæmorrhage on the outer part of which it lay. It is by no means uncommon to find that the blood from an aneurism on the surface of the brain lacerates the cerebral substance instead of escaping in what would seem the ready channel beneath the arachnoid. In a case which came under my notice some time ago the blood from an aneurism of the posterior cerebral artery, near its origin, entered the crus and passed into the substance of the pons before escaping on the surface. Probably two causes produce this result. Outside some aneurisms there is much thickening of tissue. This is especially the case in those which seem to result from imperfect embolism, which would lead to local arteritis. Secondly, it is probable that when aneurisms thus rupture into the brain, instead of the subarachnoid space in which they lie, the blood escapes from them slowly, and a slow disintegration of brain tissue opens up a ready path for the blood. The slow disintegrating character of the effusion into the brain affords an explanation of the absence of marked apoplectiform symptoms at the onset of the hæmorrhage in this and in similar cases. Apoplexy in cerebral hæmorrhage seems related especially to the shock to the

brain which results from the sudden laceration of the tissue. In slow disintegrating laceration it may reasonably be expected to be less intense. In this case there was loss of consciousness during only twelve hours before death, while during the preceding forty-eight hours there had been severe convulsions, consciousness in the intervals being unimpaired.

The peculiar ventricular hæmorrhage affords proof of the gradual extravasation of the blood. Each ventricle (except the fifth) contained clot, but each contained only a very small quantity, lying in that part of the cavity in which the least resistance would be met with. It appears to have slowly trickled along the spaces, but not to have exercised the least distending force. The fact that a larger quantity was found in the fourth than in the other ventricles may be probably ascribed to the effect of gravitation and the ease with which the ventricle, on account of its shape, might be filled.

The convulsions which occurred towards the close may have been due either to the uræmia or to the fresh cerebral hæmorrhage. There were no preceding symptoms of uræmia, and it is not improbable that the slow escape of blood into the cerebral substance was the immediate cause of the fits, although the renal mischief may have predisposed to their occurrence.

The peculiar facial paralysis deserves a special mention. I have never seen so marked a difference between the emotional and voluntary movements as in this case. It is of interest, in connection with the circumstance that the hæmorrhage was in the midst of the locality to which, in apes, Ferrier localises the movements of the face. The paralysis of the face was a motor symptom similar to the phenomenon sometimes seen in aphasia, in which, as Dr. Hughlings Jackson has often pointed out, a patient may be unable to utter, voluntarily, an expression, while he has considerable emotional (or automatic) use of language. *March 16th*, 1876.

II.—DISEASES, ETC., OF THE ORGANS OF RESPIRATION.

1. *A case of enteric fever with extreme ulceration of the larynx, and but little affection of the ileum.*

By C. Hilton Fagge, M.D.

Andrew M—, æt. 33, was admitted into Guy's Hospital under the care of Dr. Pavy, October 8th, 1875. He had been a sailor for ten years; he had always been healthy, and said that he had not had syphilis. He was attacked with headache and shivering seventeen or eighteen days before his admission, and three or four days afterwards he began to suffer from diarrhœa, which continued up to the time when he came into the hospital. He was at sea when attacked; the ship came into the port of London two days before he was admitted. The food and water on board were said to be good. None of his shipmates were attacked in the same way.

He appeared a healthy, well-nourished man. He complained of feeling generally ill; his tongue had a brownish dry fur in its centre; his fauces were covered with a thick mucus; he complained of his throat being sore; his voice was husky; his abdomen was somewhat distended; in the right iliac fossa there was gurgling. Seven or eight rose spots were noticeable on the left side of his chest. The heart sounds were healthy; bronchitic râles were audible over the lungs.

Temp. 104·5° F. . Pulse 128 . Resp. 28.

9th.—He takes his milk well; his bowels have been open once. His tongue is fissured in the centre, with a brown fur. His pulse is very feeble. Wine ℥iv.

Morning temp.	103°	.	Pulse	116	.	Resp.	36.
3 p.m.	„ 102·7°	.	„	120	.	„	32.
Evening	„ 103°	.	„	128	.	„	26.

10th.—Bowels open once. Fæces thin, yellowish, watery; spots still visible; tongue very dry. Wine ℥viij.

Morning temp. 102·7°	.	Pulse 120	. Resp. 38.
4 p.m. „ 103·8°	.	„ 136	. „ 48.

11th.—Bowels open once.

Morning temp. 104·6°	.	Pulse 124	. Resp. 52.
4 p.m. „ 103°	.	...	...
Evening „ 104°	.	„ 128	. „ 60.

12th.—Last night the bowels were open at 2 a.m. ; the spots have faded, and others have taken their place ; he passed a very restless night ; the fæces were fluid, yellowish, with pale lumps.

Morning temp. 103·8°	.	Pulse 132	. Resp. 64.
Evening „ 104°	.	„ 126	. „ 57.

13th.—Bowels open last night ; he was very restless, wanting to sit up every few minutes. Tongue very foul, but moist.

Morning temp. 102·4°	.	Pulse 128	. Resp. 56.
Evening „ 102·4°	.	„ 144	. „ 64.

14th.—Bowels open last night ; no blood in the evacuations, nor has there been on any former day ; cough very troublesome.

Morning temp. 102·4°	.	Pulse 144	. Resp. 60.
Evening „ 102·4°	.	„ 128	. „ 60.

15th.—Passed a much more quiet night ; breathing and cough better ; abdomen still much distended, with spots on right side ; bowels open last night ; no blood.

Morning temp. 101·7°	.	Pulse 128	. Resp. 52.
Evening „ 103°	.	„ 130	. „ 47.

16th.—Passed a good night, and had to be awakened for nourishment ; bowels open this morning ; evacuation not loose, and containing no blood.

Morning temp. 102°	.	Pulse 144	. Resp. 52.
Evening „ 101·2°	.	„ 134	. „ 52.

17th.—Last night he was very restless ; bowels open once.

Morning temp. 100·2°	.	Pulse 144	. Resp. 62.
Evening „ 101°	.	„ 144	. „ 60.

18th.—Passed a quieter night ; he is perspiring profusely ; takes his nourishment well.

Morning temp. 100°	.	Pulse 108	. Resp. 60.
Evening „ 101·4°	.	„ 180	. „ 56.

19th.—Passed a quieter night, sleeping well; he only once attempted to get out. This morning at 6.30 the nurse noticed that his breathing was bad, and that he had great difficulty in swallowing; but in the night he had taken three pints of milk, three eggs, and ten ounces of brandy. The house-physician was sent for at 8.30 a.m., and ordered him some Spir. Amm. Aromat. and brandy; he took a dose of this without difficulty, but died about three minutes afterwards. His bowels had been open once in the night.

A *post-mortem* examination was made the same day.

The body was emaciated in a moderate degree.

The brain was healthy.

Both lungs were very emphysematous, but there was no congestion even of their posterior lobes. The large bronchial tubes were reddened, their mucous membrane was swollen, and they contained much puriform mucus; the smaller tubes were not reddened and contained only a little frothy mucous fluid.

The larynx was ulcerated to a remarkable extent. Over the right arytenoid cartilage there was a deep ulcer, with a small surface and thick opaque, white edges, which certainly looked chronic. A probe passed into this went for nearly half an inch into the ary-epiglottidian fold, in front of the arytænoid cartilage. The fold itself was much swollen. Along each vocal cord there was a linear ulcer, with opaque white margin. Each edge of the epiglottis was ulcerated, a thin line of erosion running along it; and on the right side this contained a narrow yellow slough.

The heart was healthy; the femoral veins were free from thrombus.

The whole length of the intestine was most carefully examined, but scarcely any indications of disease could be detected. About a foot above the ileo-cæcal valve there was an ill-defined purplish-red patch of about the size of a shilling, and a little higher up there was another patch presenting similar characters, except that in its centre there was a darker spot the size of a split pea. When examined under water, this showed distinctly a breach of surface.

Several of the mesenteric glands were much swollen and very soft, and of a reddish-purple colour.

The spleen weighed six ounces; it was neither enlarged nor softened.

The liver was healthy. The gall-bladder was small, and contained only colourless mucus, mixed with some opaque flocculent puriform

material. The cystic duct was perfectly unstained with bile, whereas the hepatic ducts contained bile.

The kidneys were congested, and weighed 14 ounces.

The adductor muscles of the thighs did not, to the naked eye, present any change, but under the microscope several of their fibres were found to be enlarged to one and a half times their normal diameter, and their substance was glassy and fractured into little square masses. *October 19th*, 1875.

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2. *Syphilitic* (?) *pneumonia.*

By W. S. Greenfield, M.D.

The piece of lung and the sections exhibited were removed from the body of a female child, 12 months old, who died in the out-patient room of St. Thomas's Hospital, in February, 1873. The child was said to have had a cough for some time, and to have been ailing, but had had no severe illness. The mother brought the child to the Hospital, thinking it only slightly ill, but it died in her arms whilst waiting. No satisfactory history was obtained, and there was no distinct evidence of syphilis, but there were circumstances in the family history which rendered its existence extremely probable.

At the *post-mortem*, twenty-four hours after death, by Dr. Payne, the left lung was found slightly collapsed at the base, otherwise apparently normal. The liver was quite healthy in appearance, and there was nothing noteworthy in the other organs, with the exception of the right lung. This was completely consolidated throughout, and in a state of full expansion, with slight recent pleurisy, mainly over the lower lobe. No thickening of pleura, but a few loose adhesions of older date. The bronchi of right lung were injected and contained some muco-purulent fluid.

On section of the lung it was found pretty completely consolidated throughout, and of a somewhat yellowish or yellowish-white colour, the cut surface being smooth and slightly shining, differing markedly from ordinary grey hepatization of acute pneumonia. The tissue, very firm and tough, exuded but very scanty turbid fluid on scraping or squeezing. On looking closely at the cut surface

it was seen that minute bands of fibrous tissue ran everywhere through it.

Portions of the upper and lower lobe were kept for microscopic examination. It is to be regretted that none of the left lung was kept for comparison. The condition of the bronchial glands is not noted.

Microscopical examination of sections from the right lung (*vide* Pl. III, figs. 1, 2, 3).—On examining with a low power, the lung is found to be everywhere traversed by bands of fibrous tissue of varying thickness, running in all directions, enclosing groups of alveoli. They are, for the most part, by their branching and reunion, disposed in such a manner as to divide the lung tissue into rounded spaces, which vary in size, some being of the width of seven or eight air-cells, others somewhat larger; these spaces being for the most part irregularly rounded or polygonal. The fibrous septa vary from ·25 to ·6 of a millimètre in thickness, and the spaces enclosed are from ·25 to 1 millimètre in width. The fibrous bands are for the most part composed of a highly vascular tissue, which in some of the smaller bundles consists mainly of an imperfectly developed cell-growth. In the spaces left are some air-cells, with more or less thickened walls, in some places scarcely any alveoli being visible, owing to the great thickening and consequent compression and distortion. The thickening, under a low power, appears to be in part due to cell infiltration, in part to delicate bands of connective tissue running into the alveolar walls.

Everywhere the walls of the vessels and bronchi present great thickening, either fibrous or corpuscular; here and there they are surrounded by a large aggregation of cells; in other places they are nearly obliterated by the great thickening. The newly formed fibrous tissue is, however, highly vascular, containing large vessels, many of which are evidently of new formation.

For the most part, the remains of the air-cells show little sign of inflammation; but in some places, where the walls are less thickened, they contain fibrinous exudation and some catarrhal cells, this condition being almost limited to the lower lobe of the lung.

On examining with a higher power, the walls of the alveoli appear remarkably thickened, and the epithelium everywhere persistent and apparently increased in amount. In some places the thickening appears to be due solely to the overgrowth of the epithelium, the cells of which are perfectly normal in appearance, entirely free from signs of degeneration. The greater part of the thickening, however, is due to a nucleated cell growth, of which the cells are for the most

DESCRIPTION OF PLATE III.

Figs. 1, 2, and 3 illustrate Dr. Greenfield's specimen of Syphilitic (?) Pneumonia. (Page 43.) From drawings by himself.

FIG. 1. A rough sketch under a low power, to show the arrangement of the fibrous septa and the thickening and partial obliteration of the walls of the air-cells.

2. Shows the margin of one of the fibrous septa, with bands of fibrous tissue running between the alveoli, the walls of which are somewhat thickened, but the epithelium is normal. Hartnack, oc. 3, object. 8.

3. Here, on the right side, is seen a mass of small-celled infiltration surrounding an artery and bronchus, and possibly a vein. On the left side this infiltration is seen extending into the walls of the air-cells, which are much thickened, the cavity of some of these being partly filled with degenerated catarrhal or exudation products. Hartnack, oc. 3, object. 4.

Figs. 4 and 5 illustrate Dr. Greenfield's case of Lymphadenoma of the Skin. (Page 275.) From drawings by himself.

FIG. 4. A section from deeper layers of cutis, showing characters of new growth. Hartnack, oc. 1, object. 8.

5. A section from superficial portion of nodule, showing the mode of infiltration of the new growth. Hartnack, oc. 1, object. 4.

Fig. 1

Fig. 4

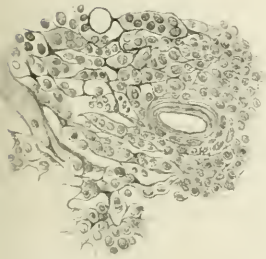


Fig. 3

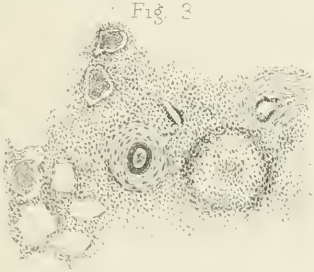
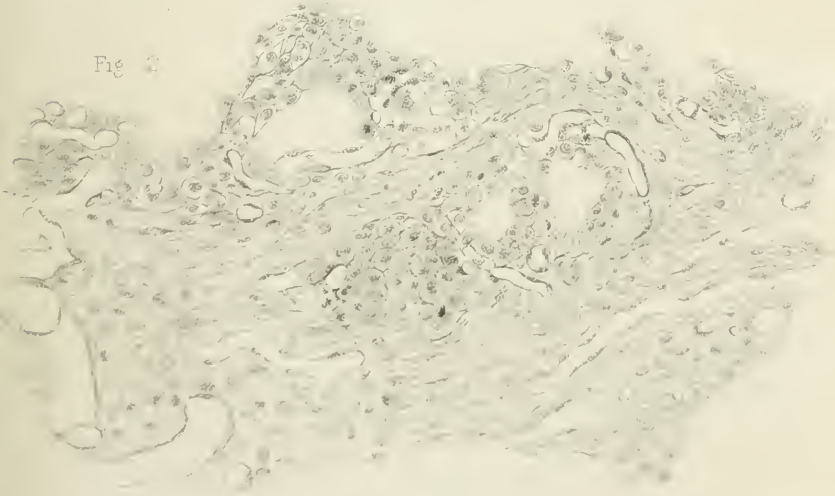


Fig. 5

Fig. 2



part elongated or fusiform, around the pulmonic capillaries, which latter appear at the same time to increase in size and to develope in some cases into larger vessels. In some places the growth appears to take place by a modification of the epithelial cells or of the capillary nuclei, but it cannot be stated with certainty that this is the case.

The alveolar epithelium is seen to persist even where the cavity of the air-cell is completely filled up, and its size greatly diminished.

Remarks.—Although I have no desire to press unduly the syphilitic origin of the disease in this case in the absence of direct evidence on the question of etiology, I may point out that it corresponds precisely in every particular with the disease of the lungs occurring in new-born or young syphilitic children, which has been described by Wagner as the diffused syphilitic form, by Lorain and Robin as "epithelioma" of the lungs, and by Virchow and Weber as "white hepatization" of the lungs. For a description of this condition, and for further references, I may refer to Dr. Wilson Fox's article on the subject in 'Reynolds' System of Medicine,' vol. iii. p. 794. It will be readily seen that the term "epithelioma" of the lung might be applied to this case on the ground of the remarkable persistence and hypertrophy of the alveolar epithelium, whilst the name white hepatization equally describes the naked-eye character. A perusal of the descriptions given by the above-named observers will, I think, place it beyond doubt that the condition of the lung in the present case, whatever its etiology, corresponds entirely with that found in the cases whose syphilitic origin was beyond question. Again, the condition of the lung and its microscopic appearances differed *in toto* from those found in ordinary chronic pneumonia and in fibroid phthisis; a comparison of sections from lungs affected with these diseases showed that there was no resemblance between the two. It is hoped that the drawings, however imperfect, will show this without a more detailed description (*vide* Pl. III, figs 1, 2, 3). Considering also the facts that the disease, of whatever nature, was of long standing, probably dating at least as far back as the time of birth, that it was entirely unilateral, of slow and of progressive course, unattended by any marked symptoms, that there was no sign of chronic bronchial catarrh, or of tuberculosis, and that the growth presents precisely the characters of syphilitic infiltration in other organs, it must, I think, be conceded as almost beyond doubt that such was its nature.

I may add that I have had an opportunity of comparing the sections

with sections from a lung affected with syphilitic infiltration in an infant, whose other organs contained gummata, and that as regards the earlier stages of the disease they correspond precisely with the latter in their microscopic characters.

November 2nd, 1875.

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3. *Obstructed lympathic glands as a cause of malignant pleuritis.*

By W. Moxon, M.D.

In the session of the Society for 1872-3 I brought forward, at different times, three cases from which it appeared that a local disease had been originated, or intensified, through the influence of obstruction in the lymph-stream by anciently destroyed lymphatic glands. These cases are in the 24th volume of the Society's 'Transactions,' pp. 20, 28, 116.

The following case indicates a like obstruction to the lymphatic stream as having caused the death of a man æt. 39. My proposition is that through an old destruction of numerous glands at the root of the lung, the nutrition of the pleural texture was so lowered by bad drainage, that on the occasion of an attack of acute pleurisy the inflammation became excessive and purulent, and the lymph-stream was invaded by pus, which also found its way into the blood; so that the patient sank rapidly with symptoms of purulent infection.

The patient, a well-developed and well-nourished man, of temperate habits, æt. 39, was captain of a merchant ship trading to the West Indies. There was nothing remarkable in his family history. He had formerly suffered severe hardships during life in the Australian "Bush." He had been twice stabbed in the arm, and once shot. He also, nine years ago, had fallen when on board ship, and it was said that he had fractured "all his left ribs." For the last seven years he had been trading to the West Indies, and during that time he had been rather severely dyspeptic, suffering constant thirst, and vomiting more or less, until he acquired great facility in the act, and could bring it about voluntarily. For several years, too,

he had been annoyed with irritability of the bladder, having to rise about five or six times every night to micturate. He had also been subject to cough and profuse perspirations, and in the course of his tropical experiences he had had several attacks of dysentery.

Two years ago Mr. Batteson, of Bow, to whose kindness I am indebted for most of these particulars, operated on him for fistula in ano, and removed some piles.

Last July, while going down Channel, he vomited large quantities of blood, and was obliged to put into Plymouth for medical aid. In the continuation of his voyage he had two fits. December 18th, he called on Mr. Batteson on his return from the Indies. He was then cheerful and said he was in good health. But on the 21st December Mr. Batteson was urgently sent for to see him and found him in bed, breathing sixty in the minute, pulse 160, and very weak. Heart sounds inaudible, mind not clear, drowsy but capable of being roused, when he spoke quite sensibly and with good memory and knowledge of things around. He lay on his back without desiring to be raised, his face was flushed and bloated, the pupils rather contracted. Temperature was 101·5. No albumen in urine, but large deposit of urates, and the urine very scanty. His complaint was of great pain in the right hypochondrium, and there was tenderness over that region and over the lower part of the right thorax. This part was moderately dull posteriorly, and the breath-sounds there were much diminished; considerable soft moist crackling was audible as high as the angle of the scapula. The left base was healthy. The heart's sounds continued almost entirely inaudible. Digitalis and brandy were prescribed, but he died in the morning of the 23rd December, having been forty-eight hours ill. During the last thirty hours he had constant vomiting and hiccough. Before death he had three convulsions at intervals.

The inspection was made ten hours after death, and yet blood decomposition was advancing rapidly, so that the interior of the blood-vessels was deeply stained, and the lining membrane of the trachea was of a deep purple hue; small blebs of gas were appearing in the blood in some parts, and there was the peculiar rancid offensive odour which accompanies this early decomposition of the blood.

But on careful search of the body throughout we could find no other definite localised morbid condition than the following, which it will be seen was limited to the right pleura; this contained about three ounces of brownish puriform liquid, evidently discoloured by

blood pigment; the left pleura was healthy. On removing the lung its appearance was at once noticed as remarkable through the presence of a quantity of yellow lines wandering upon it. These lines were lymphatics of the pleura, and on cutting any of them across pus came from them freely, so that usually a little yellow stream of pus oozed to $\frac{1}{10}$ of an inch from the cut end. This injection of the lymphatic vessels extended over the whole of the outer and hinder parts of the lower lobe, but did not reach the upper. It was remarkable that no similar disorder of the lymphatics under the costal pleura could be found. Section of the lung substance did not show any pneumonic or other changes except that the texture was slightly œdematous.

There was very little extension of the purulent lymphangitis into the lymphatics of the interlobular septa. On examining the roots of the lungs there was evidence of extensive ancient disease of the lympathic glands of the right lung. We found the right bronchus puckered within, and in two large puckered patches the surface was of sooty blackness. Incision at these spots revealed a blackened remainder of a lymphatic gland closely adherent to these old bronchial scars, these conditions having no doubt arisen from former bronchial abscesses which had burst into the bronchus. Besides these old patches of diseased glands there were several others in the root of the right lung in a similar state, and the pus streaks under the pleura could be traced close up to these relics of glands.

The remainder of the *lungs* was healthy. The *heart* was very flabby, and its interior was stained as in early decomposition of the blood. The blood in all the heart's cavities was fluid.

The *liver* was fatty to a very considerable degree, but was not unnaturally adherent to parts around.

The *stomach* was rather dilated, but its mucous surface showed no ulcer nor was any scar or trace of former ulcer discoverable.

The *bladder* was natural in all parts except that there was injection of small vessels, and a granular state of the mucous surface around the opening of the urethra.

The portion of the lung I can show to the Society is unfortunately not all I could wish, as the examination was made under the eye of a relative placed to watch the proceedings. The facts will be judged according to their merits by the Society; but this case, with the others I have alluded to, appears to me to show an effect of old lymphatic glandular disease which deserves to be brought to light.

As we have now three examples of a curious penetration of pus into the lymphatic vessels of the pleura, in cases of severe purulent pleurisy, associated in each case with an old obstruction of the glands corresponding to the affected part, I think it becomes highly probable that the prior glandular obstruction induced that severity of the inflammation which showed itself in its purulent character and in invasion of the lymphatics. To suppose a parallel case: if the glands of the axilla were destroyed, the textures of the arm would then suffer from the imperfect lymph-drainage. Then if erysipelas should attack the limb, there is great probability that the course of the erysipelas would be exceptionally severe.

January 18th, 1876.

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4. *Ulceration of the larynx.*

By Lennox Browne and Gilbart Smith, M.D.

I first saw the patient, whose larynx I now exhibit to the Society, on July 18th of last year, in consultation with Dr. Gilbart Smith. She was a pale, delicate girl of 15, tall for her age, of slight build, but not thin or emaciated. She complained of difficulty of breathing, so severe as to prevent her lying down, and of loss of voice. Her history, which I gathered from Dr. Gilbart Smith, under whose care she had been for a month, was that she had never been strong, that she had, in common with other members of the same family, been the subject of enlarged tonsils, which in her case had been removed when nine years of age. Three years previously she had suffered from a sore on the right shin-bone, which had discharged and healed, had reopened and had again healed, and that since the sore had closed about a year ago she had first begun to suffer from loss of voice.

In April, 1875, she had been seized with great difficulty of breathing, and the doctor who had seen her had said "she was suffering from severe croup." The attack lasted for a week, and left her very prostrate. In spite of change of air and restoration of general health she had not since recovered her voice. When first seen by Dr. Gilbart Smith he found the voice a mere whisper, while the breathing was loudly stridulous, both in inspiration and expiration.

She complained of a feeling of pulsation at the right side of the larynx, and also just above the sternum on the same side.

On external examination there were found several enlarged glands lying over the right carotid artery. On auscultation of the chest nothing was noticeable, except that over the right apex the respiratory murmur was somewhat feeble, and that the heart-sounds were abnormally distinct. Cough, of a well-marked laryngeal character, was very troublesome and frequent. Difficulty of breathing was constant, and was occasionally attended with spasm.

There was neither dysphagia, hectic, thirst, dyspepsia, diarrhœa, nor other constitutional symptom of phthisis. *Examination* with the laryngoscope—a process somewhat difficult from the fact that the epiglottis hung very low—showed swelling of the right ventricular band, which obscured the right true cord, but there was seen a long chain of irregular non-pedunculated growths occupying the whole of that cord. On inspiration, the right side of the larynx was less abducted than the left. The left cord appeared healthy ; there was some general thickening of the mucous membrane, but not of the epiglottis.

My opinion, given at the time, was that the case was one of growths in the larynx, with such an amount of general chronic inflammation, and occurring in a patient of such a weak and scrofulous constitution that no operative measures could be recommended. I ordered a spray inhalation of chloride of zinc with internal administration of iodide of iron and cod-liver oil. Under this treatment she became much better, the voice became stronger, the cough left her. Her appetite improved, but the breathing continued short, and was occasionally spasmodic.

On the 6th of January I received a visit from her with her mother, who stated that she had been in much the same condition until about three weeks before, when swallowing had become painful, so that she had taken less food, but that she had no rigors, night-sweats, or diarrhœa. A week previously she had complained of pain and stitch in the right side, which had been promptly relieved by a mustard and linseed poultice. The breathing had in the last few days become much worse, and it was on this account that further advice was now sought. Laryngoscopic examination showed considerable œdema of the glottis, but there did not appear any marked increase in the growth. The right side of the larynx was seen, however, to be completely paralysed. There were pains on that side on external

pressure. There was no swelling externally or sign of inflammation. I did not offer much hope from treatment, but suggested the advisability of tracheotomy to relieve the dyspnœa, caused chiefly, as I believed and expressed myself, by pressure on the right inferior laryngeal nerve, though it was, doubtless, aggravated by the mechanical obstruction of the growth and by the œdema. Dr. Gilbart Smith agreeing with me in the step I proposed, consent was obtained and arrangements were made to open the trachea on the following day. The patient returned home and slept well, but about half-past seven the next morning died suddenly and quietly in her sleep, while her mother, who was in the same room, was dressing. Her account is that the breathing suddenly became slow. She tried to waken the child, but failed, and then almost immediately she was dead, without choking or gurgling.

Post-mortem examination, twelve hours after death, was confined to the larynx, lungs, and heart. On opening the larynx the mucous membrane was seen to be generally thickened and œdematous. The whole of the right cord was eaten away by a deep ulcer, which had involved both the arytenoid cartilage and the cricoid, which latter was only covered on its anterior aspect by a very thin layer of perichondrium. On the under surface of the epiglottis there were several small punctured ulcerations. There was a large mass of more or less pedunculated soft growths occupying the right side of the larynx above and below the ulceration, and on the left side below the vocal cord. The trachea and larger bronchi were singularly free from mucus, and but slightly congested. The pleura of the right side was adherent by quite recent deposit, and rather thickened at the apex, at which point the smaller bronchi were slightly blocked with mucus. Both lungs were otherwise quite healthy, and in no way unduly engorged. The heart was in every respect healthy, and its cavities free from clots.

On searching for glandular enlargement the right bronchus was seen to be embedded in a mass of hypertrophied gland-tissue, which extended upwards more or less along the whole right side of the windpipe. It was most difficult to dissect out the pneumogastric and recurrent nerve of this side, and in doing so much of this gland-structure was necessarily removed. On tracing the nerve up to the larynx, the *right* posterior crico-arytenoid was found to be quite wasted and very pale. With the microscope it was seen to have undergone the ordinary fatty degeneration of atrophy. The *left*

muscle was of good colour and well nourished. There were a few enlarged glands on the left side of the trachea, but the nerve of that side was quite free from any pressure in its whole course.

The interesting points of this case are many, and of greater moment than can well be dwelt on on the present occasion.

Probably the case will be shortly discussed by some as one of laryngeal phthisis; but, setting aside the fact that, although I have often seen evidences of this disease with the laryngoscope before it could be detected with the stethoscope, I have never seen a case in which, after death, there has not been extensive pulmonary disease. There never were, at any time, any of the ordinary constitutional symptoms of syphilis. The lungs were examined with the microscope, and revealed no disease. It is more probable that the case was one of scrofulous disease of the cartilages of the larynx, and that the so-called growths are of the nature of irregular hypertrophy of the lining membrane, the result of inflammatory and ulcerative processes. Cursory examination with the microscope failed to show the presence of tubercle. To me the great point of interest is the pressure on the right inferior laryngeal nerve, and the consequent paralysis of the adductor of that side of the larynx. It is, I think, the first case exhibited at this Society in which the right recurrent nerve has been to such an extent involved. Of course many cases are known of similar pressure from aneurysmal and other tumours involving the left nerve, and of cancer involving the right.

April 4th, 1876.

Report of the Committee on Morbid Growths on Mr. Lennox Browne's case of ulceration of the larynx.—We have examined microscopically sections of the border of the ulcer and of the pedunculated masses on its surface. We find all parts infiltrated with leucocytes, but no new growth of any kind. The microscopic appearances presented are common to many forms of ulceration, but are not characteristic of any one form in particular. Sections of the enlarged glands presented the appearances met with in ordinary chronic enlargement of lymphatic glands.

The naked-eye appearances do not seem to us to be specially characteristic of tubercle or of syphilis.

Henry T. Butlin.
Rickman J. Godlee.

5. *Two cases of obsolete pyæmic abscesses in the lungs.*

By C. Hilton Fagge, M.D.

The specimens were taken from two children who had been in the same ward of Guy's Hospital, and whose bodies were examined on the same day in the *post-mortem* theatre.

One of them, a boy, æt. 9, was suffering from hip disease, with a foul discharging abscess. He was admitted into the hospital nineteen days before his death. He had a high temperature and a very quick pulse. I found recent pyæmic abscesses in one kidney and in the left lung. In the right lung there were two superficial spots of old disease, one on the posterior surface of the lower lobe, the other in the upper lobe. The lower one was partially calcified, and the pulmonary tissue around it was puckered.

The other patient, a girl, æt. 10, had had disease of the left knee-joint for three months, and had been in the hospital six weeks, before her death. Softening thrombus was found in the femoral and iliac veins, and even in the lower part of the inferior vena cava. There were two recent pyæmic abscesses in one lung. In the posterior part of the right lower lobe there was a cheesy patch, which I thought was clearly an obsolete pyæmic abscess. It came flat up to the surface of the organ, which was slightly puckered at that spot. The cheesy material could be turned out from it, leaving a distinct lining membrane to the space in which it lay.

May 2nd, 1876.

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6. *Croup, secondary to hooping-cough.*

By William Squire, M.D.

The larynx is that of a girl (Jane Foster), $1\frac{1}{4}$ years old, who died of croupal symptoms after nearly a week's illness. I only saw the child before noon, April 5th, 1876, on the day of her death, when the symptoms were too urgent to admit of any relief but from tracheotomy, and too extreme for me to hold out much hope from that. The child died early in the afternoon. The examination was made at noon the next day. In the trachea were detached

shreds of yellow viscid matter, one or two pieces were removed during the *post-mortem* examination, but some not quite detached were *in sitû* where they had been exuded, and some had partly descended from the larynx. The interior of the larynx was covered with exudation. This ended by an abrupt, well-defined line at the margin of the epiglottis, and by two equally well-marked tufts at the extremities of the arytenoid folds. The pharynx was injected and held some mucosities, but no deposit was seen; the posterior nares were not examined. There was only slight enlargement of the lymphatic glands at the angle of the jaw, and none of those along the larynx and trachea. The absence of such glandular enlargement was the chief post-mortem character to negative diphtheria; the general reasons against it were the presence of only croupal symptoms, and those described to me as of short duration, and the general healthiness of other children in the house. There were two families of children residing in the house, which had old brick drains and an ill-placed closet; the dust-bins were in a neglected state. Scarlet fever broke out amongst the inmates last autumn and this child then had that complaint favorably. Last February she had hooping-cough with others in the house. She was attended by Mr. Prince, the surgeon to the St. George's Dispensary, for this illness till the morning of March 28th, when she seemed very much better. The same evening she is reported to have had some signs of croup, but they were not very serious till April 3rd, when Mr. Prince was sent for. She had no throat symptoms, but the mother, who had latterly been subject to sore throat, was then complaining of it and had small spots on the tonsils. The laryngeal signs in the child were those of obstruction and not of spasm. Next day the cry was suppressed, very little air entered the lung, and the epigastrium was retracted during the inspiratory efforts; there were paroxysms of extreme dyspnœa. On the following day these were less violent, but the lips and finger-tips were bluish, and had it been possible to arrange for tracheotomy before noon, some temporary relief might have been obtained.

She died soon after one o'clock p.m.

I confess that, at first sight, I took this for a case of simple croup. I knew it was secondary to hooping-cough, but there was nothing in the general symptoms to approximate it to diphtheria rather than to laryngo-tracheitis, as a complication of hooping-cough. The circumstances that do associate it with diphtheritic cases, or to the allied forms of low inflammation, are the insidious approach of the

symptoms, and their duration. It is rarely that we do not find inflammatory croup at its worst by the third day; this did not even excite alarm till the fifth. Then, the child, instead of being exposed to cold, had not been taken out of the house, hardly out of the room, and we have evidence that the air of both was close and unwholesome. The microscopical appearances will be more fully detailed by Dr. Greenfield, who has kindly investigated them for me. They correspond as closely as any case of croup can do to what is observed in diphtheria. The loose exudation is chiefly corpuscular as in croup, but the deposit on the larynx is like what is found in diphtheria.

I wish to observe that the character of the pathological product is not so much determined by the specific cause of the inflammation as by the anatomical structure of the part inflamed. We generally find the closely attached exudation in the larynx, the loosely hanging shreds in the trachea. The former is necessarily so identified with the mucous membrane as soon as the epithelium is lost that it would not be surprising (should a patient live long enough) to find erosion of its structure as in diphtheria; that erosion is more likely to occur in diphtheria, and is more often found, is deeper and more extensive, even invading the cartilage, I have myself seen and pointed out as a circumstance differentiating it from croup, but I would rather found the distinction between the two diseases on their general history than on a single point in the *post-mortem* appearances.

The influence of season in determining the incidence of croup has again been brought under my notice by the occurrence during the past month, with its sudden changes of temperature, of three other cases. Two of them were secondary to measles; one had tracheotomy performed with good hopes of success, but was ultimately fatal; the other recovered after a prolonged and severe illness. Croup set in two days after the full rash of measles; capillary bronchitis followed the expulsion of false membrane on the fourth day, four days after that there were symptoms of meningeal irritation with a temperature of 105° F., which was again reached once before convalescence; the whole illness lasted three weeks. In this case I can be sure that there was no question of either diphtheria or of defective sanitary conditions. The whole house had been put in thorough order, with ventilation of the sewers and closets, a year before. Two other children in the family went through the

attack of measles safely and without complication at the same time; this child had previously suffered from croupy attacks.

The next specimen I have to exhibit is from a house dog who was the companion of these children and used to accompany them in their walks. He was a black retriever, five years old, who had been in the family three years. He had the distemper when a year and a half old, and was five weeks ill with it; he never ailed anything during the three years he had been in the house. From the 30th of March to the 11th of April the dog had not been near the children; on that day one of the convalescents saw him, he was brought up into the sick room, when the last and most severe case had only just passed the crisis, and was in the fullest of the rash; the dog licked the sick child's hand, and was some time in the room; he may have come into the room on the 12th, but certainly not later, for on that evening the child was seized with croup and there was too much anxiety from illness for the dog to be thought of. On April 23rd the dog was noticed to be ill, he did not take his food; next day he was taken out of doors but was soon tired; on the 25th and 26th the eyes are noticed to be red, so that they looked like those of a bloodhound; on the latter day there was much defluxion from one of them. On the morning of the 27th he was dying, with great distress of breathing and much sanious mucus flowing from the mouth and nostrils. The illness lasted four days, and came on after an interval of ten days for incubation. This is so precisely the history of measles that I was not surprised to find no obstruction in the bowels (as the groom and coachman expected who attended on the dog), but was hardly prepared to see such a close resemblance to the specific congestion produced by measles in the mucous membrane of the throat and air-passages; the dark prune-juice colour of this and of the abundant mucous secretion was very striking. What is most distinctive of measles is the character of the congestion as it now appears, with rounded raised spots, each consisting of a congeries of vessels, giving rise to projecting papillæ in the trachea, and to a mottling of the under surface of the epiglottis with a striking resemblance to the eruption of measles. The lymphatic glands of the neck were much enlarged and of a dark purple colour; the subcutaneous vessels were congested, but thick curly hair prevented any spots being seen on the skin, if any existed. I think this case proves that dogs can have measles, as it has already been proved that they can have diphtheria; it is also conclusive to

my mind that distemper in dogs in its more usual form is not measles.

Microscopic examination of larynx and trachea in Dr. Squire's case of croup, by Dr. Greenfield.—The false membrane in the *trachea* separated very readily from the subjacent tissues in a layer of about $\frac{1}{2}$ a millimètre in thickness. It was somewhat brittle. Examined when in a recent state by simple dilaceration in glycerine and water, it was found to consist almost solely of corpuscular elements and granular matter. The majority of the corpuscles were rounded, in shape and size resembling pus-corpuscles, and mingled with them were a few red blood-corpuscles. In addition, there were some large granular cells with two or three nuclei, and some scarcely altered or more or less swollen and granular columnar cells from the air-passages. Together with this there was a good deal of granular material surrounding the cells, and some colonies of micrococci. Here and there some fibrillated reticular structure was to be seen.

The *larynx* was examined, after hardening slowly in Müller's fluid, by means of sections through the whole of the structures with the adherent false membrane, the part principally examined being near the upper half of the larynx. Sections stained by picrocarminate of ammonia were mounted, some in damar, others in glycerine, and other unstained sections in glycerine.

The false membrane formed a nearly continuous layer through the whole of the larynx and on the posterior surface of the epiglottis, measuring on an average less than half a millimètre in thickness.

The greater part of the false membrane consisted of a finely reticulated structure, closely resembling a section of sponge or coral, and composed of a highly refractile homogeneous substance. The meshes of the reticulum averaged in width that of a white blood-corpuscle, their trabeculæ on an average were about half the width of the spaces, but some were wider, and the spaces were correspondingly small. In the thinnest sections most of the spaces in the network were empty, but some contained a small corpuscle. In some places the more superficial parts shewed only a very indistinct network, being composed mainly of degenerated cells and granular matter, with an imperfectly laminated arrangement.

The false membrane was everywhere firmly adherent to the subjacent basement membrane of the mucous membrane, where the latter was persistent, except where it was slightly separated at pretty regular intervals, which appeared to correspond with the

orifices of gland ducts; the spaces thus formed being filled with large corpuscles of various shape.

In some places the outline of the mucous membrane was entirely lost, the false membrane apparently infiltrating it, and the subjacent tissue or matter being continuous with a mass of infiltrated tissue.

The vessels of the mucous membrane were everywhere much dilated, and here and there the surrounding tissues infiltrated with leucocytes. The false membrane extended into some of the gland ducts. Some of the racemose mucous glands were almost unaltered, whilst others were filled with catarrhal cells. The cartilages appeared to be entirely unaffected; but there was some general infiltration of the soft tissues of the larynx.

May 2nd, 1876.

III.—DISEASES, ETC., OF THE ORGANS OF CIRCULATION.

1. *Case of aortic valvular disease, probably originating in malformation, &c.*

By Thomas B. Peacock, M.D.

M. A—, æt. 11, admitted into Alice Ward, St. Thomas's Hospital, on the 19th of March, 1875.

She was stated to have been quite a healthy child till she was seven or eight years old. She then had measles, but it was only a slight attack, and she recovered favorably and continued well for a year, when, apparently from having taken cold, she began to suffer from cough and expectoration, with shortness of breath, especially on exertion. These symptoms gradually increased, and she became seriously ill about a year before she was admitted into the hospital. She had never had scarlet fever or rheumatic fever, but at the time when her symptoms became more severe, her limbs were slightly swollen and painful, so that she could not walk, the swelling involving both feet and ankles.

Her father is a healthy man and has never had any illness, but her mother died of consumption when about thirty years of age, six years ago. She is the only girl in the family, but she has had three brothers. Two of these are living and healthy, but one of them has had an abscess in the leg, followed by chorea, ascribed to having been frightened by a dog. The third brother died when six years old of some chest affection. When admitted into St. Thomas's she had been worse for two or three months, suffering from shortness of breath, increased on exertion, and occasional attacks of sickness and vomiting. She had a pallid, puffy appearance, and the abdomen was tumid, but there was no decided dropsy. The tongue was clean and moist; the pulse 112, somewhat feeble and jerky; the respiration 32, hurried and irregular. The action of the heart was tumul-

tuous, and visible over a large space. There was decided prominence of the præcordial region. The dulness on percussion commenced in the second interspace, and became entire in the third, and extended from somewhat to the right side of the sternum to beyond the line of the nipple. There was a systolic murmur heard to the right of the upper part of the sternum of a short rough character, and this was followed by a soft diastolic murmur, which was more prolonged, and was propagated down the course of the sternum. Towards the apex there was a very feeble murmur, of a creaky character, which was evidently distinct from the murmurs heard at the base. It was thought to precede the systole, and was not audible behind, but there was no distinct purring tremor to be felt. Sibilant and sonorous rhonchi were heard in all parts of the chest. The hepatic dulness was increased in extent, and the liver could be felt extending a considerable distance below the ribs. The urine was not albuminous, and was of natural specific gravity. For the first two months of her stay in the hospital she continued much in the same state. The sick attacks occurred at intervals, and consisted of violent retching, not coming on after food was taken and indeed independently of the food or any other obvious cause. She was always very torpid and sleepy, taking no notice of what passed around her, and being indisposed to speak unless when aroused. Her temperature also was uniformly low, not exceeding 96° or 97° F.

At the beginning of June she took cold and the bronchitic symptoms were aggravated, with great increase of the difficulty of breathing and troublesome cough and expectoration. Towards the end of the month dropsical symptoms also appeared, the ankles becoming swollen and the abdomen more tumid. The urine was very scanty and contained albumen, though its specific gravity continued high, 1020; the sick attacks had, however, become less frequent and severe. The dropsical symptoms increased, and on the 20th of August she was tapped, and 9½ pints of fluid were removed from the abdominal cavity. The operation afforded much relief to the dyspnœa, but the fluid rapidly accumulated, and on the 21st of September the tapping was repeated, and 10 pints of fluid were evacuated. The relief was, however, only temporary; the symptoms steadily increased, and she died on the 10th of October.

On *post-mortem* examination, which was made by Dr. Greenfield, some fluid was found in the pleural sacs, and the abdomen was much distended. Both lungs were somewhat compressed and œdematous,

The liver presented marked hepatic venous congestion, and weighed 59 oz. The spleen was firm, and weighed 4½ oz. The kidneys were enlarged, and weighed together 8 oz. The capsules were slightly adherent, but the surface was smooth. The cortical portion was somewhat narrow, and the whole organs were dark coloured and firm, presenting the appearance of chronic induration from congestion. The alimentary canal was healthy. The reflected pericardium was attached by firm adhesions over the right ventricle and the auricle, to the beginning of the pulmonary artery and aorta. The heart was altogether enlarged and its substance firm. It weighed when opened and deprived of coagulum 20¾ oz. avoir. The walls of the right ventricle were somewhat thick. The lining membrane of the left auricle and ventricle was opaque and thick; and the folds of the mitral valve were also thickened and opaque, and the aperture was slightly diminished in capacity. The aortic valves were much thickened, and the segments were only two in number, one of them being formed by the blending together of two imperfect valves, as indicated by the existence of a raphé in the upper or arterial side. The two valves were of nearly equal size, but in consequence of the want of support at the point of union, or of imperfect separation, the edge of the united segments fell below the plane of the other valve, and so had allowed of regurgitation from the artery into the ventricle.

The segments blended together were the representatives of the right and posterior valves.

The dimensions of the heart were as follows:

Girth of right ventricle, 79 Paris lines = 177·75 mm. or 701 Eng. inches.
„ left „ 63 „ = 141·75 „ 5·59 „

Thickness of walls of right ventricle—Base, 3 Paris lines = 6·75 mm., 26 E. in. Mid-point, 2·5 Paris lines = 5·62 mm. ·22 Eng in. Apex, 2 Paris lines = 4·5 mm. ·17 Eng. in.

Thickness of walls of left ventricle—Base, 5·5 Paris lines, 12·37 mm. ·48 En. in. Mid-point, 7·5 Paris lines = 16·87 mm. ·66 Eng. in. Apex, 4 Paris lines = 9 mm. ·35 Eng. in.

Length of cavity of right ventricle—51 Paris lines = 114·75 mm. 4·52 En. inches.

Length of cavity of left ventricle—45 Paris lines = 101·25 mm. 3·99 En. in.

The right auriculo-ventricular aperture admitted a ball measuring in circumference 45 Paris lines = 101·25 mm. 3·99 En. inches.

Pulmonic aperture—39 Paris lines = 87·75 mm. 3·46 En. inches.

Left auriculo-ventricular aperture—Barely 33 Paris lines = 74·25 mm. En. inches.

Aortic aperture—30 Paris lines = 67·75 mm. 2·66 En. inches.

1. The symptoms observed during life in this case corresponded with the condition of the heart when examined after death. When first seen the systolic murmur heard in the course of the aorta, and the diastolic murmur propagated down the ventricle, indicated the existence of obstruction to the flow of blood into the aorta and its regurgitation from the artery into the ventricle, and for these signs the condition of the aortic valves afforded a satisfactory explanation. From the existence of another murmur, heard most distinctly between the nipple and the lower end of the sternum, which was thought to precede the contraction of the ventricle and was not heard in the dorsal region behind, it was supposed that there was also some obstruction to the flow of blood from the left auricle into the ventricle, but this inference was less clear from there being no distinct purring tremor to be felt towards the apex. It will, however, be seen that the mitral valve was thickened, and the orifice somewhat smaller than it should have been, when compared with the size of the right apertures and the great general enlargement of the heart. During the latter period of the child's illness the heart was displaced upwards from the accumulation of fluid in the abdomen, and the sounds of the heart could not be clearly analysed. The weight of the heart, $20\frac{3}{4}$ ozs., was at least four times what it should have been for the age and sex of the subject; a degree of enlargement which, if it had arisen within the three or four years of actual illness, was certainly very remarkable and especially so as occurring in a young person.

2. The attacks of sickness under which the child laboured were very peculiar, and their cause was not satisfactorily ascertained. They did not appear to have any reference to the time at which the food was taken or to the kind of food, and I believe she never vomited her food, but they were violent attacks of retching and vomiting. At first they occurred frequently, every two or three days, but latterly they became more rare, and were also less severe. They were present before there was any albumen in the urine, and there was nothing in the state of the stomach when examined after death to explain them. They must therefore be looked upon as spasmodic attacks due to reflected irritation from the heart. I have known the same symptom occur in other cases of heart disease, and, in a case of mitral disease, a violent attack of sickness and vomiting was the immediate cause of death, the chordæ tendineæ attached to one of the fleshy columns being torn across in the paroxysm.

3. A third peculiar feature in the case was the torpid state of the child during the whole of her residence in the hospital and the remarkable lowness of her temperature. Mr. Rossiter, the house-physician, remarked that she was always sleeping when he was in the ward, and the lowness of temperature was constant. At first it never exceeded 97·4°, and was sometimes as low as 96·8°. In June it varied between 98·5° and 95·6°, rarely, however, reaching the normal standard; and there was no difference after the evidence of kidney disease had appeared and to the last period of life, except that it rose a few days before being tapped on the first occasion, on the 22nd of August, and declined a few days afterwards. On the 18th it was 98·2° and on the 19th 99·6°; on the 20th, 98·3° and 21st 98·5°, and 22nd 98·7°, and on the 23rd 98·3°. After the second tapping on the 21st it was 99·5°, on the 22nd 96°, on the 23rd, 96°, 24th, 97·4° and 25th, 100°. In the last weeks of her life the temperature twice attained 98·4°, but was otherwise between 96·7° and 95·2°.

4. The most interesting feature of the case, and the reason which has induced me to bring it before the Society, is, however, the decided example which I conceive it forms of disease of the aortic valves originating in malformation. It will be seen that there are only two valves at the aortic orifice, and one of these is formed by the blending together, or the imperfect separation, of two segments, of which the indication is afforded by the existence of a raphé, or, as it has been termed by John Hunter, "a crossbar," on the arterial side. The blending is so complete that the two valves are of nearly equal size, and there is thickening of both segments, but not more of the united curtain than of the sound valve. The mode in which this condition originated may be regarded as open to three explanations:

1st. The union of the segments may be supposed to have been caused by the tearing down of their angles of attachment, without the curtain being involved in the laceration.

2nd. It may be ascribed to inflammation involving the two segments and leading to their adhesion; or,

3rdly and lastly. The condition may be regarded as a malformation, by which is to be understood either an improper original formation of the segments, or the union of segments, previously distinct, during intra-uterine life.

In respect to the first of these supposed causes it must be

remarked that laceration of the aortic valves is the result of violent exertion, and is attended by very marked and severe symptoms, and these persist till the fatal event, which generally soon ensues, or after from a few days or months to four or five years. Nothing at all similar can be traced in this case; though the symptoms commenced four or five years before death, they were only slight at first and gradually advanced, and had any injury been sustained it is quite impossible that it should not have been known to the parents. We may, therefore, very safely dismiss this as a possible cause of the condition.

2nd. The union of the segments can scarcely be supposed to have been the result of inflammation and adhesion of the valves, unless such illness had occurred at a very distant period, for the blending is too complete to have been of recent date; yet careful inquiry has failed to elicit any history of illness prior to the occurrence of measles, three or four years before the death of the child, and after that attack there was another period of health, lasting a year, before the symptoms of cardiac disease commenced. It is true that the child had swelling of the feet, which may have been rheumatic, but this occurred when she was labouring under cardiac symptoms, and within a year of the fatal termination of her illness. It is probable, however, that it may have been at this time that the pericardium was inflamed, as indicated by the partial adhesions found at the base and on the right side.

If also the union of the valves was due to inflammation it is curious that there should be so little thickening of the curtain affected, and that the two curtains should be so equally involved in the disease which does exist. I cannot, therefore, come to any other conclusion than that this explanation of the mode of union of the valves is at least very improbable.

3rd. In favour of the supposition that the union is a congenital defect, it may be argued—

1st. That conditions precisely similar are not unfrequently found in the hearts of children that survive birth for only a very short time. Cases of this kind have been exhibited at the Pathological Society by Mr. Obre and Dr. Quain. I have in my museum the heart of an infant which lived only six weeks, and I have seen the condition in a fœtal heart.

2nd. That they occur in persons of more advanced ages, young children, middle-aged persons, and the aged, who were never known

to have sustained any serious injury, or to have had any severe illness, and who have been entirely free from cardiac symptoms, and have been killed or have died from causes unconnected with the heart. The specimen which I exhibit, and which affords a very characteristic example of blending of two of the valves, was removed from the body of a boy, aged 15, who was killed.

3rd. They are of frequent occurrence in cases in which there are other undoubted forms of malformation. Another specimen which I exhibit to the Society affords an instance in point. Two of the aortic valves are blended together in connection with congenital contraction of the aorta distal to the left subclavian artery and an open state of the ductus arteriosus. It is, indeed, an example of that form of defect in which the descending aorta is said partly to derive its origin from the ductus arteriosus. The special interest of the case in reference to the present discussion is that the child only lived ten weeks.

The blending of the aortic valves less frequently coexists with other malformations than the similar change in the pulmonic valves, yet the two forms must be regarded as identical. I could easily have brought before the Society many instances of the latter class, in some of which there are changes which show that the deviation from the natural process of development had occurred at very early periods of fœtal life.

Taking all these circumstances into consideration, I think I am warranted in concluding, that, in this particular case, the fusion of the valves was a congenital defect. I would, however, wish to guard against being supposed to maintain that all such cases are malformations. On the contrary, I have no doubt that union of the valves does occur from disease in after life. In each particular instance, therefore, the condition of the valves and the symptoms and history of the case during life must be carefully considered before deciding as to the probable mode of origin of the disease.

If, however, in any case the condition of the valves be regarded as congenital, there may still be difference of opinion as to the mode in which the defect is produced. It may be supposed that the valves were originally well-formed, but that two of the segments subsequently became united from disease in intra-uterine life; or that the condition is a primary malformation. We do not at all know the way in which the semilunar valves are formed. When first my

attention was directed to the subject, it occurred to me that each segment might be composed of two halves blended together, the line of union being indicated by the corpora arantii in the centre of the free edge. This idea was suggested by noticing the peculiar arrangement of the semilunar folds on each side of the central muscular columns in the cartilaginous fishes, of which there are some very beautiful specimens in the Museum of the Royal College of Surgeons. I thought that the folds on each side of the central column might gradually approximate and then unite, the intervening column being finally atrophied and disappearing. This view explains both the cases of redundancy and deficiency in the number of the valves. The former being supposed to be simply due to arrest of development; the latter resulting from the subsequent adhesion of valves which had been fully formed. In support of this view I may mention that when the segments are increased in number it is rare to find them diseased; whereas when the number is deficient, they are very generally also thickened and hardened, as if from subsequent inflammation. There is also no reason why the heart should not be the seat of inflammation during fœtal life; indeed cases showing that both endocarditis and pericarditis do occur at that period are on record.

More recently, however, I have rather inclined to the idea that both the cases of redundancy and defect are due to deviations from the natural process of evolution. This view accords with the frequent coexistence of valvular imperfection with other marked defects in the heart, some of which must have originated at the earlier periods of fœtal life. It seems probable that the valves may be formed by the folding together of the lining membrane of the ventricle and artery at the orifice of the vessel, and the subsequent looping up of the band so formed into separate portions. There are cases on record in which this simple fold has been the only trace of any valvular apparatus; and I have myself a specimen in which there are no valves at the orifice of the pulmonary artery, but only such a fold combined with a band of muscular tissue. It is evident that if the supposed process of looping up may fail entirely, it may also fail to a less degree or in one part, and thus we should have the condition which is so frequently found at the pulmonic orifice, in conjunction with undoubted malformations, in which there are only two valves, with a more or less marked raphé on the arterial side; or only one valve forming a kind of disc stretched across the orifice, with three bands on the upper surface separating an equal number

of deeper or shallower sacs. The distinctness of the raphes and the depth of the sacs probably indicate the extent of the arrest or comparatively remote period at which it occurs. This supposition, however, while it more readily explains the diminution in the number of the segments, is less applicable to the cases of redundancy.

Whatever be the mode in which the defect in the number of the valves is produced, I believe that it may very greatly interfere with their functions. If there be only two segments incompetency is very apt to ensue from the want of support to the edge of the defective valve, allowing it to fall or become everted, or by the growth of the valve being interfered with in the seat of the raphé, so that the edge is, as it were, held back, and an opening is left when the orifice is closed and the valves should come in contact. On the other hand, when the three segments are united together, there is necessarily obstruction to the flow of blood from the ventricle into the artery, and often incompetency also. In both cases the defective valves very generally become the seat of subsequent disease, giving rise to thickening and induration, and often, especially when all the valves are united, ultimately causing very great obstruction.

October 19*th*, 1875.

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2. *Thrombosis of internal carotid arteries; hemiplegia; death.*

By T. S. Dowse, M.D.

M. J—, æt. 66, was admitted into the Central London Sick Asylum, Highgate, suffering from debility, on August 23rd, 1875. She died October 2nd, 1875. She had exophthalmos and arcus senilis of the cornea; her gait was tottering and her intellect dull. There spirations were normal, and the heart sounds free from bruit, but this organ gave evidence of structural change and dilatation. The urine was free from albumen.

Whilst walking down the ward she suddenly lost the use of the right side incompletely. She was conveyed to bed, became dull and lethargic. On the succeeding morning the following note was made:—Is in a state of half consciousness. She tries to answer ques-

tions, but gives evidence of amnesic aphasia. There is no marked palsy of the face either to sensation or motion. The right arm is quite powerless. The right leg partially so. There is both hyperalgia and hyperesthesia of these limbs.

In ten days after the above note was made (the palsy of the right side continuing the same), it was observed that she suddenly lost power of the (left) half of the body, the intellect became still more confused, and in a short time she sank into profound coma, and terminated her existence in about thirty-six hours.

Post-mortem twenty-four hours after death. When the brain was removed with its membranes and placed upon the table, its appearance was striking and unusual, from the fact that the anterior half of the right hemisphere was intensely hyper-vascular, and of a deep pink colour, which presented a marked contrast to the surrounding brain tissue.

Upon slicing off the right hemispheres and exposing the lateral ventricles, the same condition was observable. There was no marked convolutional change except that in which the centrum ovale participated.

The anterior lobe and the posterior as far back as the inferior parietal lobule, were of a dull red or dark pink colour from blood staining evidently of recent date, for there was no appearance of ochreous change, neither had softening taken place, although the cerebral substance was becoming plastic and granular. The vessels were dilated and engorged with blood, but none had given way except in the centre of the corpus striatum, where there was a mass of extravasation stellate in outline.

The left hemisphere was remarkably pale, and upon section gave evidence of primary, but not of advanced softening. This was not general, but confined to the centrum ovale, and grey matter in close contiguity with the posterior parts of the first, second, and third frontal convolutions. The central ganglia on this side were quite healthy. There was chronic arachnitis, with excess of fluid over the hemispheres and within the ventricles.

Upon examining the base of the brain the arteries were found to be more or less atheromatous, and plugging was especially sought after.

It was found to slightly occlude to a very short distance the middle cerebral arteries, but the other vessels were free.

Upon examining the base of the skull the carotids were seen to

be completely occluded. The petro-cavernous portion of each vessel was removed, and showed a thrombosis of each side adherent to the inner wall.

The pathology of the brain is in this case extremely interesting in relation to the vascular supply. On each side we have the main trunks plugged. On the right there was extensive congestion with extravastion of the colouring matter of the blood. On the left the brain was pale and the vessels empty.

How is this to be explained? The hemiplegia of the right side was gradual, while the left was sudden, and so far it bears out the following statement in Wilks and Moxon's 'Pathology,' when referring to white and red softening of the brain:—"We believe the difference is due to the difference in degree and rate of obstruction of the vessels. The softening is white when the obstruction is slow, so as to starve the tissue without inducing absolute stasis of the blood and subsequent hyperæmia." *October 19th*, 1875.

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3. *Case of syphilitic heart.*

By A. Pearce Gould.

The heart was removed from a labouring man, æt. 40, who fell down dead in a public house after walking in some fields, and apparently in good health. At the *post-mortem* examination Dr. Rawlins—to whom I am indebted for the specimen—found extensive apoplexy of the left lung, and with that exception the heart was the only diseased organ in the body.

On examination the anterior wall of the right ventricle was found to be composed of a greyish-white tissue, presenting to the naked eye very much the appearance of visceral cancer; it was about the normal thickness; no trace of muscle could be seen in it. This tissue was found to be extending up into the right auricle, and into the septum ventriculorum; many of the musculi pectinati were entirely composed of it. The left side of the heart and all the valves were quite healthy.

On microscopic examination I find at the edge of the new growth that there is an infiltration of small round cells between the muscular fibres; that further in these cells become more numerous and gradually replace the muscular tissue; and in the middle of the growth a thin section shows nothing but an accumulation of these cells with a good deal of very delicate fibre tissue between the individual cells, and granular débris.

For some time previous to his death the man had complained of dyspnœa and severe pain in the cardiac region, but I regret to state that I have been unable to obtain any detailed history of the man, or learn whether he had suffered from the ordinary forms of syphilitic disease.

November 2nd, 1875.

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4. *A case of embolism of the pulmonary artery in the fourth week of enteric fever.*

By C. Hilton Fagge, M.D.

Dr. M—, æt. 37, who was staying at the Inns of Court Hotel, first sent for Dr. Mumford, of Fetter Lane, on October 10th, 1874. He was said to have had diarrhœa for two or three weeks. His pulse was 120, his temperature 106°. He was evidently suffering from enteric fever, and had violent delirium for several days. Sir W. Jenner saw him on October 11th and October 17th. After this date he improved: and on the 26th his temperature was normal and his pulse 84. He was perfectly sensible.

On the 17th Dr. Mumford was sent for at six a.m., and found that in the course of the night he had insisted on getting out of bed, when he threw himself back upon the bed, became immediately excited, and said he felt worse. Dr. Mumford found his temperature 102°; his heart's action was very irregular. He complained of slight dyspnœa. His upper extremities were cold.

At midday Dr. Mumford saw him again. He was then suffering from urgent dyspnœa, and evidently was sinking fast. All the limbs were as cold as ice, and bathed in perspiration. The heart's action

was very tumultuous and irregular. Embolism of the pulmonary artery was diagnosed.

I assisted Dr. Mumford in making a *post-mortem* examination on October 28th.

Head not examined.

Lungs healthy: their anterior edges somewhat emphysematous. No pneumonia or hypostatic engorgement. Bronchial tubes reddened, containing much mucus.

Heart flabby: its muscular tissue pale and soft. The valves healthy. The right auricle and ventricle containing gelatinous clots, in great part decolorised, entangled among the chordæ of the tricuspid valve. The base of the pulmonary artery had in it black blood. The bifurcation of the pulmonary artery, and its two main divisions, contained a horizontal bar of firm *ante-mortem* clot. This was non-adherent, except that it sent prolongations into most of the primary divisions of the vessels in each lung. It had a very remarkable appearance: it seemed to be made up of four distinct rounded cords, each of the diameter of an ordinary lead pencil, which were closely applied to one another, and were twisted round one another, and to some extent interlaced. After the clot had been floated out in water, we found that these cords, apparently distinct, were really parts of a single straight piece of cylindrical coagulum, which had been folded on itself. The prolongations into the secondary branches of the artery were not continuous with this principal mass, but were superimposed upon it, so that they could easily have been picked off without breaking its continuity. No marks of valves could be seen on the clot.

The vena cava, and the iliac and femoral veins were all empty, or contained *post-mortem* coagula.

The liver was large, soft, and probably fatty.

The spleen was large and soft.

The cæcum and ileum contained several of the characteristic ulcers of enteric fever; these were clean, with smooth rounded edges; they had shed their sloughs. They would correspond with the fourth week of the disease.

Some of the mesenteric glands were suppurating.

The kidneys were much altered by decomposition, but apparently healthy.

Remarks.—I think this case is of interest, not only on account of the peculiar way in which the clot obstructing the pulmonary

artery was folded on itself, but also because of the completeness with which it had been washed out of the vein in which it had been formed, leaving no fragments to indicate its original seat. There can be little doubt that it had arisen in one or other of the femoral veins; for not only is thrombosis there very common in convalescence from enteric fever, but probably no other vein in the body could contain a clot so large and of so great a length.

November 16*th*, 1875.

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5. *Cured hydatid cyst in the wall of the heart.*

By James F. Goodhart, M.D.

The specimen was removed from a young man æt. 20, who fell down dead in a stable in Portman Market. Mr. Hodson Rugg was summoned to him and made the *post-mortem* examination. The man had been dancing and singing half an hour previously, and at the time of his death was engaged in pulling sheep into a stable by the legs—a somewhat laborious occupation.

His mother stated that she had never known her son ill at any time, but on closer interrogation she stated that he had suffered from slight achings of the limbs for which he had attended St. Mary's Hospital as an out-patient twelve months ago. He was an inmate of the Clerkenwell prison for kicking a horse from January to March, 1875, and he told his friends that he fell off the wheel two or three times from exhaustion. This was considered to be mere laziness on his part, and he was punished accordingly by being placed in the "blackhole." Latterly he had been noticed to be a very heavy sleeper, and his friends had considerable difficulty in rousing him by shaking. He had probably had syphilis.

At the *post-mortem* thirty-six hours after death, extensive extravasation was found on the surface of the brain, but the arteries were in good condition. The pia mater also showed patches of recent lymph. The lungs were congested.

Heart.—Pericardium adherent, and in dissecting it back the point of the scalpel entered an abscess in the anterior wall of the

heart, from which flowed three to four ounces of pus accompanied by some fibrous material. The other viscera were healthy.

The specimen is a cured hydatid cyst occupying the anterior wall of the left ventricle and the septum between the ventricles. It is about the size of a Tangerine orange and full of pultaceous hydatid membrane. Large numbers of fat crystals, granular matter, and not a few hooklets and fragments of hooklets were seen by the microscope. The inner wall of the left ventricle shows a thick whitish hard mass which forms part of the wall of the cyst beneath the endocardium. The heart is of about normal size and the cavities and valves normal. The muscle of the left ventricle is perhaps rather thin. The pericardium was very much thickened and adherent over the front of the heart—less thick but still adherent behind.

Of the seven other cases previously recorded in the Transactions only two occurred without known symptoms of heart or lung disease before death and in both of these the hydatid was still in a living state. The interest of the case lies in the fact that the cyst was quite shrunken, and therefore much smaller now than it had been, and yet no serious symptoms appear to have ever been complained of. From the quantity of pultaceous hydatid membrane found in the cyst after death it may indeed be inferred that the cyst must at one time have been very large ; from the amount of fibroid material around it is probable that it was also of considerable standing yet the patient would seem to have completely recovered and died subsequently by the accident as it were of suppuration round the old cyst. It may, however, have been that the suppuration occurred and killed the hydatid in which case its life may have been of shorter duration than one would suppose possible, judging from the surrounding induration. *November 16th*, 1875.

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6. *Ulcerative endocarditis ; vegetations on, and perforation of, aortic valves ; aneurysm of mitral valve ; splenic infarctions.*

By Sidney Coupland, M.D.

This heart, which shows very admirably the characters and mode of formation of so-called "aneurysm" of the mitral valve, was

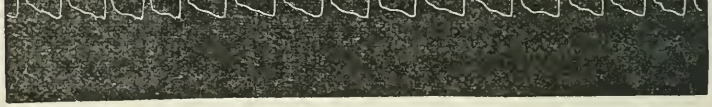
obtained from the body of a man, æt. 40, who was admitted into the Middlesex Hospital, under the care of Dr. Thompson, on November 4th, 1875. For the following notes I am indebted to Dr. Finlay, who was then resident physician's assistant.

He was unmarried, a clerk by occupation, and had never suffered from acute rheumatism himself, although one of his brothers was said to have died from that disease. There was no other rheumatic history in the family. The patient himself had occasionally suffered from pains in the knees and ankles, but never so as to confine him to bed. He had indeed enjoyed good health up to the spring of the present year, when he is said to have had an attack of "pleurisy in the left side."

About five weeks before his admission he was attacked with acute pain in the precordial region, an attack not preceded by any marked shivering, or attended by any articular pains. Almost since the commencement of the attack he has suffered severely from shortness of breath, and has been unable to lie down or make any exertion. He has been much troubled with cough, and within the last few days pain has come on in the left shoulder.

State on admission.—Face pale and earthy; tongue thickly coated; pulse sharp, thrilling, and collapsing. [Dr. Finlay took some sphygmographic tracings of the pulse, one of which is here figured, *vide* woodcut 1]. Arteries of wrist and neck tortuous and locomotive.

Woodcut 1.



Precordial dulness extends to about one and a half inches below the nipple. The heart's action is tumultuous. There is a harsh systolic murmur audible over the whole chest, front and back; at the apex for the most part obliterating the natural first sound. The second sound is fairly well heard, especially to the left of the sternum, but it is veiled at times and in places, and followed by a murmur. In the carotids the loudness of the tracheal breathing interferes with accurate auscultation; but everywhere the second sound is muffled,

murmur-like, or wanting altogether. There is slight œdema of the ankles. The finger ends are clubbed; nails markedly curved. He complains of some pain in his left shoulder, which has been constant since its accession yesterday morning.

November 8th.—No definite evidence of angina . . . . .

November 10th.—Some impairment of resonance along the right side of sternum, most marked and most extensive in the first interspace. During diastole in the carotids there is only a murmur or silence.

November 13th.—Passed a bad night; much cough and dyspnœa.

November 15th. 9.30 a.m.—Seized with an attack of dyspnœa, and died in two hours.

At the *post-mortem* examination there was found a patch of lobular hepatisation in the lower one fourth of the upper lobe of the right lung, but no evidence of infarction. Beyond this there was great pulmonary engorgement. The pericardial sac contained a few drachms of clear serum. The heart was much enlarged, weighing when empty of blood sixteen and a half ounces. On the visceral pericardium were several milky-white patches, chiefly on the anterior surface of the right ventricle and on the intrapericardial portion of the aorta. There was a fair amount of fat on the surface of the organ, the muscular tissue itself being pale and flabby. Both ventricles were relaxed and contained clots; some few tags of decolorised fibrin adhering also to the walls of the right ventricle. Tricuspid and pulmonary valves natural. The cavity of the left auricle was large; endocardium not thickened. Bulging into the chamber from the upper surface of the anterior mitral cusp was what at first view appeared to be a large mass of vegetations. On slowly injecting water through the incompetent aortic orifice the stream was seen to trickle into the auricle through the centre of the protruding mass. Closer examination showed that the protrusion consisted of an "aneurysm" of the mitral valve, it being formed by a pouch covered by the thin membrane forming the auricular surface of the valve. The pouch was sacculated, and about the size of a Barcelona nut. It was directed slightly upwards, and its upper extremity was perforated by two apertures of considerable size, from which projected some shreds of decolorised fibrin. The margin of the valve was entire and natural, and its chordæ tendineæ were of normal tenuity. Viewed from the ventricle, the right half of the anterior

mitral cusp was seen to be the seat of an ulcerated aperture, situated about half an inch from the attached and from the fore border of the cusp. This aperture could admit the tip of the little finger, and led into the pouch above described. The margins of the aperture were fringed by small vegetations; and the ventricular fold of the valve was sharply cut around this margin. Two small superficial erosions occurred in the endocardium of the ventricle on the basal line of the triangular space between the central and left aortic cusp. The left half of the anterior mitral cusp was opaque from atheromatous change. The aortic valve was extensively diseased. Each of the cusps were more or less thickly covered by warty vegetations, which were wanting only from the contiguous portions of the right and central cusps. The central cusp was, however, the most thickly covered by vegetations of the three, and was in addition perforated to the extent of two thirds. The right and left cusps were coherent and puckered. The aortic lining membrane was stippled by atheroma, and the vessel was dilated. The wall of the left ventricle measured three quarters of an inch in thickness. Liver and kidneys slightly indurated. The spleen weighed thirteen ounces, and was the seat of two wedge-shaped infarctions; one large and fleshy, two and a half inches in depth, occupying the middle third of the organ; the margin of this block being of a pale yellow colour, the other smaller, of the size of a chestnut, and of darker tint, with deep red marginal line.

With regard to this specimen I would only remark upon the presence of vegetations on the aortic valve in association with the ulceration of the mitral, and upon the erosions on the endocardium of the ventricle. It is suggestive of the mitral lesion being directly induced by the contact of the aortic vegetations with the anterior mitral cusp. There do not now exist any vegetations long enough to come into contact with this cusp at the seat of ulceration; but the presence of the splenic infarctions, especially of the larger and older one, are facts which strongly favour the presumption of some larger and freer fibrinous vegetations having been washed away subsequently to their exciting the local endocarditis, which resulted in the formation of the aneurysm.

November 16*th*, 1875.

7. *Case of aneurysm of the ascending aorta.*

By Frederick Robinson, M.D.

A private in the Scots Fusilier Guards reported himself sick, and was admitted into hospital on the morning of the 29th September, 1875. He was an old soldier, æt. 46, and had served eighteen years. His appearance was indicative of greater longevity; for although stout and erect in person his hair was quite white.

On the date referred to he complained of pain in his left side and his breathing was hurried and distressed. On examination a slight soft systolic murmur was heard over the aortic valves. The heart's sounds elsewhere were feeble and his pulse was weak but regular. No cough or other symptoms of chest affection were complained of. The man's countenance was somewhat pallid. The pain soon passed off after the application of a sinapism and the administration of a stimulant, and the next day the patient was up, going about and declaring himself to be well again. There were then no general indications pointing to the disease from which he died, and consequently, he was not examined with reference to the possible existence of aneurysm. His breathing was ordinarily composed and tranquil; he did not cough; his voice was natural; his countenance free from anxiety. He suffered from syphilis many years ago in 1862, but afterwards enjoyed good health and led a steady life.

His ailment was diagnosed as aortic valvular disease associated with atheromatous degeneration of the great vessel.

He was ordered quinine and iron, and appeared to derive benefit from the treatment.

He continued to declare himself to be in his ordinary state of health and fit for duty, and consequently, on the 12th of October, was discharged from hospital and ordered such light duty as would be suitable to his condition pending his discharge from the service as an invalid. On his way back from the hospital to the barracks he complained of faintness and his accoutrements were carried by another man. Owing to this circumstance he was kept wholly from duty, the tendency to syncope did not return, and there was nothing in the man's condition to call for his removal again to hospital. He was able to go about in fact without active or distressing symptoms of any kind.

The medical officer in barracks was sent for at midnight on the 25th of October, and found the patient dead. He had just expired, his mouth filled with frothy mucus but no blood mingled with it.

Autopsy forty hours after decease.—Body well nourished and muscular.

Head.—Normal.

Lungs.—Left, slightly congested. Right, in a state of congestion posteriorly, breaking down readily beneath the fingers.

Liver.—Healthy, but congested.

Heart.—Pericardium contained a considerable quantity of bloody serum. The organ itself enlarged and cavities dilated. The left side thickened somewhat. The left auricle enlarged to double its normal size. Its walls were thin and blood-stained. The right pulmonary veins and commencement of left were dilated to an inch in diameter.

Aorta.—Dilated, thickened and sacculated on the right side of the ascending part of the arch. About a third of an inch above the semilunar valves was an aneurysm about the size of a large walnut communicating with the vessel by an opening as large as a shilling. The walls of the sac were extremely thin posteriorly. It was filled with tolerably firm fibrinous clots somewhat decolorised. The aneurysm had pressed on the pulmonary veins behind, hence causing, apparently, congestion of the right lung. No opening could be found between the sac and pericardium.

Some thickening was observed on the edge of the mitral valve from atheromatous deposit, and patches of the same nature in an early stage were noticed in various parts of the aorta.

This case illustrates well the difficulty, perhaps I might say impracticability, of diagnosis in some forms of thoracic aneurysm.

In the present instance the symptoms warranted a belief that disease of the aortic valves, and not to an extreme degree, was the affection under which the patient suffered. In a clinical point of view the case is instructive. I do not connect this disease with degeneration of tissue from syphilis.

December 7th, 1875.

8. *Fibroid thickening of the tissues in the anterior mediastinum; obliteration of the superior cava; malformation of the pulmonary valve; fibroid disease of the heart; dilatation of the right side of the heart; chronic peritonitis.*

By S. O. Habershon, M.D.

Thomas P—, æt. 37, by occupation a coal-heaver, was admitted into Guy's Hospital, under Dr. Habershon's care in June, 1874. He had suffered from small-pox when young, but had remained well till 7 years ago, when he was suddenly attacked with swelling of the face and neck; he was, however, able to return to work on the following day. A few days afterwards the swelling returned, and he was unable to do any work for two years; from that time he had done lighter work. The patient was admitted into the hospital with swelling of the abdomen, there was much lividity of the face, and head, and œdema of the arms; when he stooped downwards, the lips, and ears became purple from congestion. The veins were enlarged, the pulse was compressible, the heart feeble, and the sounds distant, and there was considerable dulness in the clavicular region of the right side. The veins on the anterior surface of the abdomen were enlarged. The patient was kept quiet, and iodide of potassium was given continuously. There was steady improvement, and he left the hospital relieved after a stay of sixteen weeks. He was re-admitted on March 22nd, 1875; he had not been away from the hospital many days when the ankles became swollen and six weeks before admission his legs and left arm became enlarged, and of a blue colour. He stated that he had never been a hard drinker, but there was a doubtful history of syphilis. His height was 5ft. 7, and his weight 11 stones. The skin was dry and cool, but much congested. The right eye had been partially closed since birth. The ears, nose, cheeks and lips were much congested and of a purple colour. There was great distension, but no obvious pulsation of the external jugular veins, and a vein as thick as the little finger passed obliquely across the trachea, between the sterno-mastoid muscles. The supra-clavicular spaces were filled up, but were not œdematous. The abdomen was forty-two inches in circumference; the skin was smooth and tense. The swelling bulged out in either flank. In the umbilical region also the resonance

was impaired. Large tortuous veins passed downwards from the ensiform cartilage and seemed to bifurcate at the umbilicus; fluctuation could be detected in the abdomen.

There was dulness across the upper part of the chest especially on the right side; the cardiac dulness was also increased, extending three inches to the right of the median line, as high as the upper border of the second costal cartilage, and two inches below the left nipple. The apex beat was scarcely perceptible; the 1st sound of the heart could scarcely be heard; the 2nd sound was faintly audible. The lungs were apparently healthy, the vesicular murmur was feeble, but heard throughout the chest; vocal fremitus was perfect; the respiration was principally performed by the diaphragm. The ankles were œdematous, and several spots of purpura were observed on the legs. The patient complained much of flatulence, and there was occasional vomiting of frothy fluid. The pulse was feeble, irregular and compressible, 70 per minute, respiration 20. Temperature 98·6. The urine was scarcely half-a-pint in twenty-four hours, sp. gr. 1030, loaded with lithates, and slightly albuminous. It was high coloured, and free from sugar. Diuretics were given, but the dropsy and venous distension increased; the distress of the patient was very great. Paracentesis abdominis was performed, but the relief was very partial, and he sank on the 10th.

Dr. Goodhart made an inspection ten hours after death. *Brain.*—The left vertebral artery was much larger than the right. The substance of the brain was healthy. The cervical glands and the thymus and thyroid were normal. The right pleura contained about a quarter of a pint of fluid, the left pleura half a pint, but there was no inflammatory product. The lower lobe of the left lung was in an early stage of pneumonia, soft, and very œdematous. The lower lobe of the right lung was also œdematous; the upper lobes were healthy. There was a complete absence of any of that usual discoloration or toughness seen in disease of the mitral valve. The bronchial tubes were generally healthy, some contained a little purulent fluid. The larynx was healthy. On opening the body the first thing that was noticed on reflecting the skin of the neck was an enormous dilatation of two veins, running obliquely towards the middle line of the neck to the top of the sternum. These were anterior jugular branches, and were not only much dilated, but their coats were much thickened; still they were not as thick as arteries, nor were they atheromatous. That on the right side, which

was the smaller, seemed to end above the sternum in communicating with the subclavian branches, and also in the innominate vein. The left terminated in the left innominate vein. Tracing these on to the superior cava, the latter was found completely obliterated. The two innominate veins came each separately to a stop in a blind puckered pouch in some tough fibrous mediastinal tissue to the right of the aortic arch. The superior cava appeared absent altogether, and its proper seat of opening in the auricle was represented by a thickened, whitish, and smooth endocardium, much like the appearance of a closed foramen ovale. Underneath this could be felt tough gristly substance, but there was no well-defined tumour anywhere; on a cross section being made, there appeared dense fibroid tissue, and an increase of the mediastinal connective tissue. The root of the lung was quite free. The left vena innominata had an outlet in an enlarged "small" azygos vein running down by the side of the aorta. The other innominate vein had communications with the anterior mediastinal veins, and particularly with one passing down with the phrenic nerve. It also communicated with intercostal branches, and probably with a very large and tortuous vein running down behind the sternum in the median line, and opening into the right internal mammary; the latter and its fellow were not notably large. The axillary veins and the subclavians on both sides were very large, and their thoracic branches were also much dilated. The supra-scapular vein on both sides was large. The external jugular vein was in a normal state. Both subclavian veins were also plugged by ante-mortem coagula, slightly adherent to the walls of each. The azygos major was completely obliterated as it turned round the root of the lung; but on tracing it down it was found enormously dilated, and its intercostal tributaries were so much distended that the little finger could easily be passed into their orifice. It appeared that blood had got into the azygos, then into the lumbar veins, and so into the inferior vena cava. The deep epigastrics were not much enlarged. The circulation seemed to have been carried on by three channels: 1. By anastomoses of the veins of the neck, particularly the anterior jugulars with the internal mammary branches, and so down the front of the abdominal walls. 2. By regurgitation through the innominate into the subclavian veins, and so round the surface of the chest again to the epigastrics. 3rdly, and chiefly, by the small azygos communicating with the left vena innominata and the lumbar veins and cava; in a similar manner, the right azygos, which collected its blood from

the surface of the chest, also joined the lumbar veins and inferior cava. The pericardium contained a little fluid, and there were old adhesions at the base of the organ around the great vessels. There was no recent inflammation. The heart was much enlarged, especially on the right side. The wall was very thin and fibrous-looking; and on section, towards the base it was hardly recognisable as muscle, but was quite white and fibrous. This state extended to the base, and was evident in the auricle, and also in the left ventricle just below the aortic valve; the most diseased spot, however, was below the pulmonary sigmoids. These valves were in a very curious condition; there were only two of about the normal size and healthy appearance, and the space for the third remained vacant; a fold of membrane ran from one part of this unvalved space, on a somewhat lower level, to one of the other valves. This fold had no corpus Arantii or other valve characteristic, except that it was formed of a fold of endocardium. The obliteration of this valve was at the spot where the greatest amount of disease existed in the mediastinum, or rather, both the pulmonary artery and the fibrous wall of the heart were directly contiguous with the mediastinal thickening. The tricuspid was large, admitting six or seven fingers. Its edges were thin. Both auricle and ventricle were dilated and full of clot. The left ventricle appeared to form a mere appendage to the right, but it was also dilated and hypertrophied. The valves of the aorta and the mitral valve were healthy. The aorta above the valves was in a very diseased state; it was extensively atheromatous, but not calcareous. The pulmonary artery was normal. The thoracic and abdominal aorta were not much diseased.

There was thickening of the peritoneum from chronic peritonitis. The cavity was full of dark yellow ascitic fluid, sp. gr. 1020. There was also recent peritonitis in the region of the transverse colon. The cæcum had become somewhat displaced and turned upwards, so that part of the surface above the "caput cæci" had become adherent to the transverse colon; and in separating the adhesions, which were of recent nature, the bowel gave way. On opening up the cæcum its coats at this spot were very thin, and covered by a tough yellow adherent membrane of diphtheritic appearance. The other portions of the intestine were healthy. There was excess of mucus in the stomach. The liver weighed 59 oz., it was much altered in shape and rounded; the capsule was thick and marked with depressions. The substance of the gland was healthy and also the vessels. The

mesenteric glands were healthy. The spleen was very firm, with thick white patches on the surface; its weight was 5½ oz. The supra-renal capsules were healthy. The kidneys weighed 10½ oz., and were quite healthy, so also were the testicles.

Many instances of obliteration of the superior cava arise from mediastinal growths pressing upon the vessel, or from coagula within the vessel; but it is probable that in this case there was congenital defect, and that no true superior cava had ever existed. It is interesting to find that for thirty years the man followed a laborious occupation. *December 7th*, 1875.

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9. *A case of disease of the pulmonary and tricuspid valves of the heart.*

By Alexander Morison, M.B.

Acquired valvular disease of the right side of the heart, apart from incompetency due to obstruction in the left side, is so rare, and yet, as in this instance, may occur in so aggravated a form, that I have considered the case hereafter related worthy of record. Dr. Walshe,[1] speaking of systolic basic murmur, best heard at the third left cartilage, says: "If we except cases of cyanosis where stenosis of the vessel (pulmonary artery) is the most common lesion, the conditions capable of generating the murmur are so rare that few persons have actually met with an example," and he himself disclaims any *post-mortem* experience of such, as also does Dr. C. J. B. Williams.[2]

For his attempts to determine, chiefly by experiment, the physical diagnosis of valve disease in the right heart Dr. Hope is taken to task by Dr. Stokes,[3] who in a manner reprimands him for endeavouring to establish that of which he had no satisfactory and indubitable clinical experience, and the latter answers in the negative a question as to the possibility of diagnosing such disease from physical signs,

[1] 'Diseases of the Heart,' fourth edit., p. 98.

[2] 'Diseases of the Chest,' edition 1840.

[3] 'Diseases of the Heart and Aorta,' 1854.

apart from anatomical considerations. Dr. Stokes,[1] however, quotes a case of patent foramen ovale, with thickened and retracted pulmonary valves, as supplying in a measure this hiatus in clinical experience. Bichat[2] erroneously considered the pulmonary and tricuspid valves exempt from disease, as cases showing an affection of one or the other have been recorded in the remote past, as well as more recently by writers at home and abroad.

Failure to discover a case similar to that I am about to relate, either in museums or in the literature of the subject, together with the conviction that in such a case no well-ascertained signs or symptoms can be considered of little moment, must form my excuse for entering somewhat into detail.

Mr. E. W. G. M—, aged twenty years and nine months, a bank clerk, single, and of temperate habits, came under my care on May the 20th, 1875, complaining of breathlessness, both when active and when in bed, with occasional attacks of severe pain over the heart, passing up to the right shoulder and down the right arm, being particularly felt at the tip of the right little finger. He had also frequently experienced pain of a most acute and continuous character about the lower end of the sternum, which lasted on different occasions from half an hour to four or five hours, and was not influenced by food or drink. In bed he is most free from uneasiness, when lying on the right side, with the head bent forward, and should he when asleep lie on his left side the heart-pain wakens him. When lying on the back the head is turned to one side or the other. He thinks that, on the whole, he is less embarrassed when sitting up than when lying down. Complains, too, of a feeling of irritation about the nostrils, with a choking sensation, and during the last two or three months has occasionally had slight attacks of epistaxis. For a year his feet have sometimes swollen across the instep towards night, but no trace of such remains next morning. He suffers much from general weakness, and has occasionally had what he calls fainting fits. When *five* or *six* years old the patient suffered from scarlatina, which is said not to have been severe, and to have left no perceptibly evil results. As a child he had frequent attacks of croup, but there is no definite information regarding any other disease of childhood. When *eight* years of age he was examined by a medical man for admission to an educational charity, and was

[1] Op. cit., p. 166.

[2] Laennec, 'Diseases of the Chest,' translation.

found to have heart disease. At the age of twelve he ran a race, fainted, and became "cold all over." Animation was with difficulty restored, but next morning he did not appear much the worse of the accident. Except on this occasion he betrayed no sign of delicacy, and but that his relatives were aware of his having heart disease, he was considered healthy till the age of fourteen, when he left home and entered into business. Though frequently overworked and exposed to cold he continued in fair health for four years, having, however, on two or three occasions had severe "pain in the side," for which a doctor attended him. When *eighteen* years of age a medical man gave him permission to play cricket, and to this exercise he and his relations ascribe his gradual decline in health. Increasing dyspnœa and loss of strength forced him to give up work about a year ago, and since, his symptoms have progressively become more serious. He had *never* suffered from acute rheumatism.

Patient's father died insane, and his mother of consumption. Two paternal uncles and a paternal aunt died of heart disease between the ages of thirty-two and sixty. A paternal first cousin, who, with four or five brothers and sisters, has had rheumatic fever, suffers from heart disease. One maternal uncle died of heart disease, having had rheumatic fever, and another has had pneumonia. He has a brother and sister who have not had any serious illness.

The patient himself is fairly grown, but emaciated; with grey-blue eyes, light brown hair, a pale, somewhat livid, countenance, with a patch of purpura over the left malar prominence, and an anxious expression. The pulse, when lying, beats 78–84 in the minute; is *regular*, small, but distinct, and when the arm is extended from the shoulder becomes slightly weaker; both pulses are equal. There is well-marked pulsation in the supra-sternal fossa, and on pressure behind the notch a distinct systolic impulse is communicated to the fingers. This visible pulsation, though chiefly systolic, has an undulatory character, as if partly diastolic. There is distinct, but not remarkable, regurgitation into both external jugulars. The carotid pulse is *normal*, though, from the emaciation of the patient, somewhat more apparent than usual. The præcordial region to the left of the sternum is uniformly bulged forward, and the left subclavicular region is sunken, apparently owing in a measure to a somewhat greater prominence of the left than of the right clavicle. The apex beat is diffused and its impulse *weakened*. There is *visible*

systolic pulsation from the second *left* intercostal space to below the nipple, most distinct in the *second* and *third left spaces*, and again, in a limited area, immediately above the nipple, but at the latter point the heart's impulse is *not* more distinctly felt than elsewhere. There is well-marked *pulsation* in the *epigastrium*.

On laying the hand over the præcordial region, and *especially* over the second and third left spaces, a harsh *systolic* and *diastolic* "frémissement cataire" is felt. The heart's dulness is increased *transversely*, and runs with great distinctness into the liver dulness. *No* increase of dulness from above downwards was detected.

On auscultation to the *inner* side and below the left nipple, *close* to the left edge of the sternum, there is heard a distinct systolic bellows murmur, *lost* on tracing it outwards towards the left chest. On continuing to auscultate along the left edge of the sternum, a *double murmur* also becomes audible, increasing in distinctness as you trace it upwards, until, over the cartilage of the *third left rib*, and in the *second left* intercostal space, a *harsh grating systolic* and *diastolic bruit* has its position of maximum intensity. This murmur is also heard, but *less* distinctly, at a corresponding point to the right of the sternum; but over the aortic area the aortic second may, on careful observation, occasionally be heard, apparently healthy, though much masked by the coarsely grating murmur to the left. The murmur is *not* propagated into the large systemic arteries, but towards the *left*, in the course of the pulmonary artery. His respirations, when lying, count 40–42 in a minute, and there is very violent action of the nostrils, a phenomenon which seems to have come on gradually, but has been much more apparent during the last three months, and is quite as evident when the patient is in bed as it is when he is upright. The subclaviau regions expand imperfectly, and he is unable, apparently from the rapidity with which the respiratory acts follow one another, to take a deep and full inspiration. Percussion resonance generally is not markedly dull, but is high-pitched. Vocal resonance is nearly normal, though rather increased than diminished. He has a short, hard, and frequent cough, with an expectoration principally mucous, but sometimes muco-purulent. The respiratory murmur *generally* is *very harsh*, but *without* any moist sounds. He has *never* had hæmoptysis. The tongue is clean, but the papillæ at its edge near the tip are enlarged and congested. Appetite bad, flatulent eructations, but *no* vomiting. His bowels have as a rule acted normally, but of late

more frequently, with semifluid evacuations of a somewhat dark colour. The liver dulness in the mammary line measures about six inches. The spleen was not satisfactorily mapped out. Says he urinates freely, and has never had any symptoms specially referable to this system. The skin presents nothing abnormal, except the small patch of extravasation before alluded to over the left malar prominence. The special senses are healthy, he never suffers from headache, and the intellect is clear. The treatment adopted was: Rest in the recumbent position, nourishing diet, with restriction of fluid, syrup of the phosphate of iron, and 5–10 minims of the tincture of digitalis in a mixture every three to four hours. Apparently, by this means, his heart's action became slower and less excited, the supra-sternal pulsation for a time almost entirely disappeared, and when it returned did so gradually. The jugular pulse also appeared less marked, but his general distress was *not* much alleviated.

In the beginning of June he had a thickly sown eruption of purpura over the forearms, wrists, legs, and ankles, which gradually faded. Such was his condition till about the middle of June, when another and more copious eruption of purpura made its appearance in the same positions and to a less extent on the trunk, together with a feeling of weakness and pain in the lumbar region, and though the quantity of his urine remained unchanged, and was sufficient, it had a dark and smoky appearance. On June 16th it is noted to have had the following characters: "Sp. gr. 1005; acid; smoky; albuminous to about one quarter of the quantity of urine in the test tube; abundant tube casts, principally granular, with adherent renal epithelium; many of the casts are dark coloured, others more transparent; a few fragments of blood-casts; red blood-cells; renal epithelium, and crystals of uric acid." He also complained of headache, became drowsy, and had some diarrhœa. Mustard counter-irritation over the loins, with an occasional draught of sulphate of magnesia, was followed by a disappearance of the lumbar pain, and greater intelligence, but his urine continued as before and began to diminish in quantity. The reddening caused by the mustard was seen to consist of a thickly sown sprinkling of minute points of *extravasation* resembling a fine purpura, except that the points had an *arterial* hue.

June 19th.—Urine very scanty; slight diarrhœa; no vomiting; purpura gradually fading. 10 p.m. Called suddenly to see patient.

Urine almost suppressed; temperature 101·8; pulse 120; respirations 60; skin dry; lungs as before. After a purge and poulticing the loins the most urgent symptoms subsided, and the eruption continued to fade.

June 22nd.—Purpura again in larger patches than ever, especially over the *right* wrist and elbow, on which side he lies; urine still very scanty; drowsy; slight diarrhœa; pulse 104; respirations 42.

June 23rd.—Urine, sp. gr. 1005.

June 24th.—About 7 a.m. began to swell about the *right eye* (he lies on his right side); at 11.50 a.m. the right was closed with œdema, the left becoming œdematous, and the forehead and scalp up to the vertex, *especially* towards the *right* side, was tense, painful, swollen, and pitting on pressure; *no* pitting over the sternum or tibiæ; pulse 102; respiration 42; urine as before. Was ordered a purge and a diuretic mixture containing digitalis, squills, spirits of nitrous ether and Liq. Am. Acetas, after which a larger quantity of urine was voided and the œdema disappeared.

On June 27th he is noted to have had *hæmoptysis* to about one ounce, the blood being arterial and mixed with air, and the physical signs in the chest as before, with slight comparative dulness at the left base, but the extreme bases were throughout somewhat less resonant than elsewhere. The purpura was still persistent.

June 28th.—Heart's action *regular*, signs as before, pulse 108–114, *regular*, but very weak; respirations, 42; *violent* action of the alæ nasi; fæces fluid and dark; urine as before; drowsy. He died next day, having been conscious to within half an hour of death, when he apparently became unconscious and died *without any* evident increase in his distress of breathing, and *without any* sign of convulsion.

Necropsy twenty-nine hours after death.—Body much emaciated. Purpura persistent on the arms, legs, and right side of the trunk. On opening the chest the lungs seen *retracted*, their inner margins at the nearest point being fully an inch and a half apart, and exposing the pericardium over the heart and large vessels. *Pericardium* smooth and without any trace of inflammation, recent or remote, its cavity containing about an ounce of straw-coloured fluid. The parietal layer, at a point opposite the heart's apex, finely mottled from an extravasation of blood; the visceral layer also near the apex having a similar extravasation. *Heart, in situ*, increased in size, especially transversely, the margin of the right ventricle, at a

point opposite the axis of the conus arteriosus, being *very* obtusely angular. The heart weighed, after being in spirit for some days, nine ounces and five drachms. *Right auricle* dilated, with its closely packed columnæ carnæ well marked. A warty vegetation springing up into its cavity from the insertion of the anterior segment of the tricuspid valve at its inner extremity, and two others on its posterior wall, pass downwards and inwards through the auriculo-ventricular orifice, their lower extremities going to form part of a warty mass on the internal segment of the tricuspid. The broadest ends of the growths are in the *auricle*, the largest nearest the lumen, the other two between it and the wall of the auricle, that next the wall being smallest and flat.

The posterior surface of the anterior segment of the tricuspid (*i.e.*, the surface looking into the auricle in systole) is covered, especially along its margin, with warty growths. These vegetations combined encroach considerably upon the auriculo-ventricular space. *Right ventricle* is dilated and hypertrophied, but the former condition preponderates. All the segments of the *tricuspid* are studded with warty excrescences, more especially the anterior and internal segments. The former has a thickened and irregular margin, and the latter is in fact replaced by these growths. The posterior segment lying against the right wall of the cavity is also thickened by a warty deposit. The musculi papillares are somewhat hypertrophied. The *pulmonary* semilunar valves are replaced by a distorted mass of warty clumps. The segment most to the right when the artery is laid open retains its shape most perfectly, but its margin is enormously thickened, and below and on all sides of it there are excrescences. The central segment consists of an enormous mass of warty growths, a *large pear-shaped process* from which stretches into the pulmonary artery, and when unsupported hangs at right angles to its stalk, and probably occupied this position in diastole, and may *perhaps* to some extent have acted as a valvular prevention to regurgitation. Behind this segment and overlapping to some extent the segment to the right, there is a *flattened mass* of excrescences, attached to and passing into the pulmonary artery, for more than an inch, occupying in fact that portion of the arterial wall which must have been *struck* during each systole, by the mass of the *central* valve. The segment most to the left consists, likewise, of a misshapen warty growth, about an inch in length and breadth, having a faint resemblance to a sigmoid valve, and having an irregularly diamond-shaped *perforation*,

and here too a crescent of small granulations on the wall of the artery *coincides* exactly with the upper margin of this altered segment. On the anterior wall of the conus arteriosus there are scattered excrescences of various size and shape. Towards the apex of the ventricle and attached to a slender chorda tendinea there is a vegetation. The circumference of the pulmonary artery at the line of the valves is $3\frac{5}{8}$ inches, and above the valves $3\frac{6}{8}$ inches. A large partially decolourised fibrinous clot passed from the site of the pulmonary valves up to, and *into*, the two principal branches of the artery, on dividing which the clot was seen to occupy the entire lumen of each vessel. The clot was most decolourised towards the *ventricle*. A large clot of a similar nature was drawn through the auriculo-ventricular orifice, and there were other smaller clots clinging to the chorda tendinea. The clots clung most tenaciously to the growths. The excrescences have a cartilaginous or gritty centre, with a soft circumference. Microscopically they appear to consist of oval or rounded nuclei, in a granular, more or less opaque stroma, at places much impregnated by an earthy deposit. The portions examined did not present any appearance of a fibrous stroma. The sections cleared on adding dilute hydrochloric acid, and less so with acetic acid.

Left auricle normal. Mitral valves healthy, except that the shorter has two small warty deposits, about the centre of its auricular aspect. At the margin of the other segment, on the same side, there is a growth of the size and shape of a split pea.

Left ventricle normal, circumference of the aorta at the line of the valves $2\frac{1}{2}$ inches; above the valves $2\frac{3}{8}$ inches. Semilunar valves competent, and healthy. At the base of the *centre* segment there is a rounded cavity, with a minute warty excrescence at its orifice. On holding this part up to the light, the septum is seen to be thin and *translucent* (a natural condition) and a large blunt probe passed into the above-mentioned small cavity *tore through* the partition, and presented itself below the internal segment of the tricuspid valve, the point emerging in a bed of granulations, the site of a large vegetation. At the origin of the aorta there is a trace of commencing atheroma. The endocardium in the *right* ventricle, especially in the neighbourhood of warty clumps, is *thickened* and *opaque*. The coronary arteries so far as examined were healthy. Muscular fibres taken from the right cavities of the heart had well-marked cross striæ. A few enlarged glands which lay between the pul-

monary artery and aorta were dissected away, but I regret that the former vessel was not examined throughout.

The *pleuræ* were generally adherent over both lungs, presenting many old adhesions of varying length, those at the apex and base being shorter than those in the intermediate space. The cavities contained no fluid.

The *left lung* floats in water and crepitates uniformly, though not quite freely. At its extreme base it seems to be in a state of chronic consolidation. The lung generally is of a firmer consistence than is natural, and contains circumscribed patches of induration. Below the pleura, at the base posteriorly, there are a few ecchymoses similar to those in the pericardium. The mucous membrane of the bronchi was reddened and some tubes contained pus, others casts of inspissated secretion, but there was no evidence of breaking down lung tissue anywhere. *Right lung* in the same condition as the left, but rather firmer at the base, and containing in this portion a comparatively recent extravasation of blood about the size of a walnut. Air-cells in the indurated portions contained blood, but there was no evident convolution or enlargement of the vessels in the fibrous septa. Other portions examined presented microscopically healthy lung tissue.[1]

Liver.—Longest measurement of the right lobe about eight inches, of the left five inches. Organ firm to touch with a smooth capsule, and on section gross and microscopic presenting the appearances of nutmeg liver.

Spleen. Seven inches long, four inches wide, firm to touch, with a somewhat thickened capsule, a well-marked notch in its anterior border, and presenting no distinct evidence of amyloid degeneration. *Pancreas* firm, connective tissue between its lobules increased, and its veins engorged. The *left kidney*, enlarged, cortex increased, capsule peeling off readily, surface smooth, finely sprinkled with myriads of engorged Malpighian tufts, and having a few arborescent veins. The *right kidney* in its upper two thirds like the left, its lower third having a much lobulated surface and being separated from the remainder by a deep sulcus; the cortical and pyramidal portion of this part very scanty, but the former relatively increased. With the microscope many of the tubules were seen to be dilated, some bereft of epithelium and others filled with blood or their own

[1] Compare 'Text-book of Practical Medicine,' by Niemeyer, American translation, p. 359.

degenerated cells. The Malpighian tufts were intensely engorged. *Stomach.*—Mucous membrane engorged, with numerous ecchymoses, much thickened, and the follicles individually enlarged, giving the whole a *mammillated* appearance. The *small* was of a darker colour than the *large intestine,* and the mucous membrane of both passively engorged. The *omentum* contained a very small quantity of dark coloured fat. The *head* was not examined.

Remarks.—The physical signs in this case were indicative of pulmonary obstruction and regurgitation, and of tricuspid incompetency, together with dilatation or distension of the pulmonary artery. The unusual nature of the case rendered its correct diagnosis a matter of interest, and one had to discriminate between the actual state of affairs and (1) disease of the left heart, and (2) those conditions of the right heart, pulmonary artery and aorta, capable of generating physical signs more or less similar to those detailed, viz. *b.* aneurysm of the aorta; *c.* dilatation of the pulmonary artery pure and simple, without valve lesion; *d.* ossification of the pulmonary artery; *e.* aneurism of the pulmonary artery; *f. de novo* murmurs in connection with coagulation in the right heart, and thrombosis of the pulmonary artery; *g.* pressure upon the pulmonary artery by material in the pericardium; *h.* abnormal communication between the right and left sides of the heart, with stenosis of the pulmonary artery. I need scarcely say that I have had no personal experience of many of these conditions, but they have all been noted by competent observers, and I have considered the present occasion not inappropriate for giving in *outline* the principal diagnostic points between them and the case related.

(*a.*) The situation of the murmurs, and their want of propagation into the large arteries, together with the state of the radial and carotid pulses, the impaired and diffused apex beat, the site of visible pulsation, and the fact of the aortic second sound being occasionally heard apparently healthy *through* the murmur, served to exclude disease of the aortic valves. I was inclined, however, to believe that there was some *mitral* regurgitation, but the failure to follow the systolic apex bruit outwards to the left seemed to negative such a conclusion, and although the necropsy revealed slight deposit on the mitral segments, I am of opinion that the valve was competent.

When a pulmonary or tricuspid murmur is dependent upon disease of the left heart, most frequently mitral regurgitation, it may be, aggravated by aortic disease, the signs of dilatation with those

usually diagnostic of mitral and aortic valvular disease will decide the question.

(*b.*) It is possible for *pulsation* in the second and third *left* intercostal spaces, and a *bruit* in this region, to be produced by aneurysm of the *aorta*, but such an aneurysm to extend so far to the left must of necessity be sacculated, and large, and would therefore present *dulness* on percussion. "The sound also of such an aneurysm would be a low key, and as if remote, instead of loud and superficial."[1]

(*c.*) A simply dilated pulmonary artery without valve lesion, may pulsate in the second and third left intercostal spaces, and have a systolic basic bruit, but the absence of a *regurgitant* murmur, would serve to distinguish such a case from the present.[2] Dr. Hope relates one very instructive instance of this kind the signs of which led him to diagnose hypertrophy of both ventricles with "dilatation of the origin of the aorta, probably forming an aneurysmal pouch towards the left." The necropsy revealed a much dilated pulmonary artery, with stretched but *healthy* valves and a somewhat *contracted* aorta. The same author remarks that, "as the pulmonary artery is close to the surface, the sound possesses in a peculiar degree the character of proximity to the ear," and this statement I can fully endorse, from my experience of the present case, but here, in addition to the *nearness* of the vessel to the surface, the *proximity* of the sound was I believe still more marked, owing to a partially *retracted lung* as shown by the generally high-pitched percussion note, and flattening under the left clavicle, without the usual signs of breaking down lung tissue.[3]

(*d.*) A calcareous state of the wall of the pulmonary artery may cause murmur over the semilunar valves, whose competence will, however, be established in the absence of a regurgitant bruit. Such a case may also present in a marked degree (as in the present instance) the phenomena of "*air hunger*."

(*e.*) In the few recorded cases of sacculated aneurysm of the pulmonary artery there has been observed "a pulsating prominence centralising in the *second left* intercostal space, close to the sternum, a strong systolic *thrill* and systolic, *superficial*, *loud*, *harsh*, *rasping*, or *grating* murmur, of maximum force at the prominence, and followed in one instance where a pulmonary valve was contracted by

[1] 'A Treatise on Diseases of the Heart,' &c. J. Hope, M.D., &c., p. 455.

[2] Hope, op. cit., p. 594.

[3] Hope, op. cit., p. 598.

a very short *diastolic* murmur."[1] It will be observed how closely these signs resemble those described in the present case, and how with a contracted pulmonary valve, to distinguish between the two would become well nigh if not absolutely impossible, in the absence of a marked prominence. The prolongation and character of the diastolic murmur would, however, probably be influenced by the amount and kind of valve destruction.

(*f.*) Dr. Walshe[2] also states that systolic basic and infra-basic murmurs may be originated *de novo* by coagulation in the right heart and at the pulmonary orifice, and mentions the case of an old gentleman who died suddenly in the course of an acute affection from coagulation in the pulmonary artery and partly in the right ventricle, in which he heard such a murmur. A murmur thus caused independently of valve disease could of course only develop itself shortly before death, and the history or previous examination of the case should serve to distinguish it. Such a condition is most likely to arise in the aged, the much debilitated, and in puerperal women.

(*g.*) An extremely rare cause of murmur in the pulmonary semilunar region is said to be pressure upon the vessel by morbid products in the pericardium.

(*h.*) Murmurs connected with the various malformations of the heart are almost as various as the conditions which produce them, but that which interests us most at present is the occurrence of a communication between the auricles or ventricles with contraction *more* or *less* of the pulmonary artery. Under these circumstances physical signs resembling those of this case may arise, and as there need not necessarily be accompanying cyanosis, the diagnosis of this form of congenital from acquired disease in the right heart may be impossible. An illustrative example of this condition is the case of patent foramen ovale with retracted pulmonary valves (the calibre of the vessel is not stated), before referred to as quoted by Stokes, and first related to the Dublin Pathological Society by Dr. Gordon of that city. I have recently, through the courtesy of my friend Dr. Reid of Canonbury, had an opportunity of examining the heart of a girl, aged thirteen years, who is said to have had heart disease from her infancy, and is only slightly cyanosed. In this case the murmurs which were heard in the same situation, though *loud*, had none of the coarse *grating* of those in the pulmonary area of the

[1] Walshe, op. cit., p. 558.

[2] Op. cit., pp. 459 and 551.

case which I have described, and the *double* murmur at the third left cartilage became *purely systolic* on tracing it towards the *left* shoulder, while in my case, owing possibly to the retracted lung, and *gross* nature of the valve disease, together with the dilated condition of the vessel, both bruits continued to be heard. Without *post-mortem* inspection of the lesions in this case, no one would venture positively to predict either the nature or extent of the disease, further than that there was probably both obstruction at and regurgitation through the pulmonary valves, with some abnormal communications between the cavities; yet it may not be unreasonable to suppose that when murmurs present so coarse and grating a character as in my case, the valve segments are more likely to exhibit the grossly contorted appearance described, and should the case be congenital, the original malformation has in all probability been active in producing an endocarditis with further consequent changes. Whereas in a young subject with a history of heart disease from infancy, a *distinct* murmur but *free from harshness* results with greater probability from the *original* malformation, with little or no superadded disease. Impossible, however, as it may be to distinguish with any certainty between such cases, no one can fail to agree with Dr. Walshe as to how practically useful such a knowledge would be in cases of rheumatism or kidney disease, with heart complication, could the latter be diagnosed as congenital.

Well-marked as were the morbid appearances, and self-evident the agents in the production of the various physical signs in the present instance, the true etiology of the case is open to much discussion. Was the disease congenital, traumatic, due to scarlet fever, to disease of the kidneys, or finally to a combination of two or more of these possible factors, and influenced, it may be, by a rheumatic diathesis? Not to argue the point tediously, I may state shortly that, from a careful study of the entire history of the case, and of the heart itself, it appears to me that the *primary excitant* to disease was *scarlatina*, in a patient of *rheumatic* diathesis, and one of a family prone to *heart* disease; that this scarlatinal endocarditis had led to *change* in the valves, rendering them incompetent; that the *altered segments* themselves from their *disturbance* of the blood current, and *impact* against various surfaces, tended to propagate disease; that the *anæmic* condition of the patient, possibly standing in some relation to his *kidney* disease, may have aggravated matters,

and that finally *rupture* or displacement of one or more segments, perhaps a result of exercise at cricket, crowned the catastrophe, impairing still more the efficiency of an organ already labouring at great disadvantage, and precipitating the inevitable result.

I shall conclude by directing attention to a few points of interest in the case. When we consider the very serious obstruction at and regurgitation through the valves of the right heart, we cannot but be struck with the remarkable *absence* of *dropsy* into the subcutaneous tissue or serous cavities. It is feasible to suppose that when purpura appeared, the obstruction was so great as to cause that ecchymotic extravasation, and *too great* to allow of the gradual escape of the watery constituents of the blood, so as to produce anasarca, &c., but this does not explain why, at a period prior to the occurrence of purpura there was no or very trifling œdema. We can only conclude that the compensatory balance, established by hypertrophy, must have been very complete, and it is interesting to note that it was only during the *last* year of life that the patient occasionally noticed the œdema across the instep, and that it was during this year that a sensible increase of embarrassment in his condition was observed. I would also indicate the unusual *dyspnœa uninfluenced by position*, though somewhat increased by exertion, as shown by the rapid respiration and violent action of the nostrils, being almost diagnostic of his lesions, and resembling in some degree the symptoms usually ascribed to partial *embolism* or *thrombosis* of the pulmonary artery, a resemblance as *real* as it was apparent, for the huge dendritic clumps, and altered valves, filling up the mouth of the vessel, were no mean substitutes for a clot, and, though this only applies to a period shortly prior to death, the discoloured clot passing through the pulmonary orifice, along the artery and into its main bifurcations, *was* a true thrombosis, and *this* again may have increased the retraction of lung apparent on raising the sternum. The condition of the lungs seen on *post-mortem* examination was of interest in so far as they contained hæmorrhagic infarcta, and otherwise evinced the presence of hyperæmia. As the obstruction and valvular insufficiency existed at a point in the circulation before it enters the lungs, one might *à priori* have expected a less, in place of an excessive quantity of blood in those organs. The contrary is probably due to the absence of adequate *vis-à-tergo* to propel the blood through the arterial and venous radicles into the pulmonary veins, the direct cause of the inadequacy being regurgitation through

the pulmonary semilunar valves. In a case such as the present the circumscribed patches of induration may also possibly be owing to the detachment of small granulations, or portions of such, and consequent embolism of some smaller branches of the pulmonary artery. It is also worthy of notice that the *angina* the patient experienced was distinctly limited to the *right* side, as we may conclude from the fixed agonizing pain at the *lower* end of the sternum, darting up to the *right* shoulder and down the *right* arm. The tendency of the purpura and œdema of the face and scalp to gravitate towards the *right* half of the body was doubtless due to the *position* of the patient, and the appearance of the œdema in the neighbourhood of the *eye* rather than elsewhere showed its *renal* rather than cardiac origin, as was further proved by its disappearance when the flow of urine was increased, the condition of the *heart* remaining unaltered.

December 7th, 1875.

Report by the Committee on Morbid Growths on Mr. Alexander Morison's specimen of disease of the pulmonary tricuspid valves.— We have carefully examined the specimen referred to us, and find nothing to add to the very full and accurate description accompanying it. We fully agree with the author as to the nature of the case and its probable etiology, so far as an examination of the specimen permits of the formation of an opinion on the subject.

J. F. Payne,
W. S. Greenfield.

May 16th, 1876.

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10. *Aneurysm of the arch of the aorta, separating the coats of the œsophagus, and bursting into the stomach.*

By Frederick Taylor, M.D.

W. D—, æt. 53, was admitted into Guy's Hospital under my care, June 9th, 1875. He was a pipe-layer by occupation, and had been accustomed to lifting great weights, but had always until recently enjoyed excellent health, with the exception of slight

cough. He had had gonorrhœa, but never rheumatism or syphilis. He had been five months ill with a cough and a feeling of tightness across the upper part of the sternum, but he continued at his work until one month before admission, when difficulty of breathing compelled him to give it up. Since this time he had also noticed that he was unable to swallow solids, and could take only two or three mouthfuls of fluid at a time. Even these were sometimes regurgitated, exciting a hoarse cough, with discharge of more or less frothy mucus. He had, moreover, lately suffered from orthopnœa at night, and had lost flesh.

On admission he complained of pain and distress at the upper part of the chest, of shortness of breath on exertion, and of difficulty of swallowing. The food appeared to him to stop at the level of the top of the sternum, and he took only small quantities of any kind at a time. On examination of the chest there was found depression above the right clavicle, with slight dulness, hollow breathing, and marked vocal resonance; on the left side the respiratory murmur was deficient below the clavicle. The heart's apex was two inches below the nipple, its impulse slight, and the sounds clear; the precordial dulness was normal, but there was some dulness at the upper part of the sternum. The radial arteries were hard but not tortuous, equal in size and fulness. The pupils were normal, and there was nothing worthy of remark in the condition of the abdominal viscera.

On June 12th he complained of pain over the transverse colon, and on June 15th had passed a bad night on account of the same pain in addition to some cough. On the 18th the pain ceased; pulsation was observed on this day over the upper third of the sternum and corresponding intercostal spaces. On the 19th, at 4 a.m., he sat up to take some milk, and fell back dead on the pillow.

Post-mortem examination.—The stomach was distended by a large clot of blood closely moulded to its surface; there were also a few ounces of fluid blood, as well as two smaller coagula, one of which contained two masses of disintegrating fibrin. At the upper part of the stomach, near the œsophagus, was a smooth oval ulcer, about an inch long, with slightly undermined edges. Near this and to the right of the œsophagus was a rounded nipple-like projection with an aperture in the centre; on laying this open with the scissors, it was found to lead to an elongated channel filled with blood clot,

running upwards by the side of the œsophagus to one inch above the bifurcation of the trachea, where it terminated in a dilated thick-walled cavity which would have held a walnut. The rest of the canal was of larger diameter than the œsophagus and was marked throughout with transverse muscular bundles; it was obviously a channel formed in the coats of the œsophagus. At its upper extremity it communicated by an aperture, which would admit a cedar pencil, with the sac of an aneurysm of the aorta.

The aneurysm affected the third part of the arch, involving especially the concavity and the right side of the vessel, measuring six inches in circumference, and about two and a half inches in length. The aorta below it quickly narrowed to $2\frac{1}{2}$ inches in circumference; while the first part of the arch was large, measuring four inches at the sigmoid valves, and bulging opposite the great sinus. At its posterior part the aneurysm pushed its way between the trachea and œsophagus; and at a point about one inch below the origin of the left subclavian artery communicated by the aperture already described with the channel in the œsophageal wall.

There was no communication between the aneurysm and the œsophagus; but the latter was narrowed by the projection of the dilated end of the cavity above described. At this point was a small coagulum of old date, and the mucous membrane was sloughing over a space of about the size of a florin. The rest of the œsophagus, above and below, was healthy.

The lungs were very emphysematous; the bronchi were not compressed, but there was some blood extravasated beneath the pleura at the root of the lung, as well as about the diaphragm, and under the serous membrane of the stomach near the lesser curvature.

The heart weighed thirteen ounces; the aortic and mitral valves were healthy. The vertebral column was not touched by the aneurysm. The brain, liver, spleen, and kidneys were normal.

Remarks.—The above case of aneurysm is of interest as presenting an unusual mode of termination. The aneurysm occupied the last part of the arch, and pressed upon the œsophagus; but instead of bursting into its canal, as often occurs, the blood pushed its way only so far as the transverse fibrous coat, and tearing down this along the whole length of the œsophagus, broke finally into the cavity of the stomach.

This process was probably not completed in a short time, for the condition of the walls of the dilated upper end of the artificial

channel indicated that the extravasation into the walls of the œsophagus at this point was of comparatively old date, and that it only slowly extended towards the stomach was shown by the similarly thickened coat of the lower part of the channel, by the nipple-like projection into the stomach, and by the adjacent superficial ulceration.

The further progress of the blood beyond this point necessarily led to a fatal hæmorrhage.

December 21*st*, 1875.

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11. *Embolism (?) of the muscular tissue of the heart in a case of stenosis of the mitral valve, with ante-mortem coagula in left auricular appendix and embolic masses in the kidneys.*

By James F. Goodhart, M.D.

A child of fifteen was admitted to Guy's Hospital under Dr. Habershon, on November 19, 1875. She had had measles, whooping-cough, and scarlatina, but never rheumatism. Three years ago she had some cough and shortness of breath, but never dropsy till two months ago, when she was admitted into Bright Ward, under Dr. Moxon. At that time she had a large heart with both presystolic and systolic mitral bruits, a good deal of bronchitis, and also albuminuria. Under the use of digitalis she improved and left the hospital, but soon her condition deteriorated on leaving off the medicine and forsaking the quiet of hospital life.

On readmission she had much orthopnœa, continued cough, and abundant expectoration. The heart was enlarged, acting very tumultuously and irregularly, and at the rate of 148 per minute. There was a distinct thrill and presystolic bruit. The pulse was irregular and feeble, and its beats much less rapid than those of the heart. The urine contained one sixth of albumen. The liver was hardly enlarged. Under the use of digitalis she again improved, the heart becoming more steady, beating 100 per minute; it remained irregular, both presystolic and systolic bruits were subsequently heard. She got so much better as to be able to get up, but then became worse again, with much short cough and vomiting, with the

pulse rising to 150. The dyspnœa then increased, and the distress became greater and she died.

Autopsy.—The brain was not examined.

The lungs were bulky and tough, but everywhere crepitant. They were somewhat emphysematous.

The heart weighed 13 oz. The right side was very large, forming the apex, and the walls were tough and thick. On the posterior aspect of the heart, mostly over the upper part of the left ventricle, below the auriculo-ventricular ring, the muscle had a superficial yellowish discoloration, and on a section of the walls at this part the muscle was found of a dark purple colour from extravasation of blood into its substance for an area of more than a square inch. The muscle around was faded and yellow as if from fatty changes in it. The fat surrounding the coronary sinus was just below its root, abundantly spotted by ecchymoses. No embolism or diseased vessel could be found anywhere. The coronary sinus and arteries were healthy.

The right auricle was dilated, holding 4 oz. of clot. The left auricular appendix was full of a raspberry-cream-like fluid, the left auricle being dilated and its walls quite thin.

The mitral valve was contracted, its ring being tough and rigid, only admitting one finger about $1\frac{1}{4}$ inch in circumference, its edges thick. The aortic and pulmonary valves were healthy. Those of the tricuspid had a rather thick edge, but were practically healthy.

The only other viscera that need a note are the kidneys; they weighed $10\frac{1}{2}$ oz.; one had a large embolic mass in it, taking up nearly half one side. It was yellow, and with a zone of injection around it. A smaller depressed infarct occupied the other kidney; it was yellow in appearance as the other.

Under the microscope the muscular tissue of the heart in the ecchymosed parts was very much diseased. The muscular bundles were separated by a quantity of indefinitely granular material, blood-corpuscles, and also by a number of large round granulation-like cells. The muscular fibres were many of them glassy-looking or fibrous, and they constituted one of the noteworthy points in the case, for I thought I could distinctly trace a fibrous change in the muscle as the result of atrophy without any previous origin in a cellular stage.

Empyema; suppuration in the region of the tonsil; early pericarditis; extravasation of blood into the septum ventriculorum.

A girl, æt. 16, was admitted into Guy's Hospital under Dr. Wilks, in October, 1874. With previously good health she had caught a severe cold on October 4th, and since then had had a bad throat and cough. When admitted the whole of the right chest was dull, bulging, and all sounds of respiration were absent. The heart was also somewhat displaced to the left. Her temperature was 103·2° F.; pulse 136; respiration 56. The urine 1019, albuminous. The chest was tapped on the 23rd, and two pints six ounces of pus removed. The cavity was thenceforward washed out daily, and she appeared to be doing well, when on November 25th, while the chest was being washed out with a weak solution of carbolic acid as usual, and she was sitting up in bed, she turned livid, fell backwards, and ceased to breathe. Artificial respiration was commenced, and respiration by that means re-established, but she never became conscious, remaining comatose with convulsive twitchings till death.

At the *post-mortem* the brain weighed 38 oz.; it was in every respect normal as to its consistence and membranous coverings, so also was the spinal cord. No clot could be found in any of the veins.

The right lung was carnified, the pleural cavity being obliterated by adhesions and bands of lymph. The parietal layer was $\frac{7}{16}$ inch thick from granulations. There appeared to have been some old cheesy change between the right lower lobe and the pleural aspect of the diaphragm, but, excepting this, the lung was healthy though compressed. The other lung was healthy.

The heart was in the following condition, and to it I wish to draw the attention of the Society.

It was of normal size and form.

The parietal layer of the pericardium where adherent to the lung was injected, and a few flakes of lymph were noticed about the base of the heart.

The right side contained half a drachm of tough fibrinous clot, which had probably formed during the last hours of life, but which certainly was not of any date sufficient to explain the patient's manner of death. The valves on both sides were healthy. The left side contained less clot than the right.

On looking at the septum ventriculorum from its aspect towards

the cavity of the left ventricle it was seen to be of a purplish tint, as if from some dark something in the muscle. Sections of the septum showed that from the base downwards, and at the base most, its greater part was infiltrated or stained with blood. Towards the base the muscle was extensively and evenly stained, but farther down the ecchymosis limited itself to the environs of a large coronary vein. The muscle so affected was unaltered in consistence; if anything, perhaps it was rather more tough than usual.

The coronary sinus and arteries, &c., were carefully searched without finding any clot or other cause for the state found. Microscopical examination of the ecchymosed parts shows that much of the coloration is due to staining of the muscular bundles, but there is also actual extravasation of blood. The muscular bundles are many of them not good. The longitudinal striation is abnormally plain, whilst the transverse is very indistinct. This is a condition noticed in some cases where the heart muscle is becoming fibrous. Some of the muscle is very granular, but upon this too much stress must not be laid. No plug could be seen in the vessels or capillaries.

The muscles in other parts of the body showed no trace of any like ecchymosis.

The kidneys were large, 12 oz., but healthy.

A case is recorded by Dr. Hilton Fagge, where with cancer of the heart, lymphatic glands, and liver, anæmia and fatty heart were associated with meningeal apoplexy, and in this case the greater part of the right ventricle wall was intensely ecchymosed. The patient was delirious and drowsy during the last few days of her life, but it does not appear that her death was in any way attributable to the state of the heart. Two others are to be found in the 'Pathological Transactions,' in vol. ii, p. 190, and vol. x, p. 326. Of the latter it is said—the case one of diphtheria, fatty heart, renal disease, and internal hæmorrhages in a boy of ten:—

"The muscular tissue of the heart was pale, but almost all the musculi papillares, columnæ carneæ, and the walls of the apical half were almost black from nearly uniform infiltration of blood. A similar condition was present to a less extent in the right ventricle. The muscular tissue of the heart generally was in an early stage of fatty degeneration, more advanced in those portions infiltrated by blood than elsewhere."

It is not possible to associate this case with the two I have recorded, because with diphtheria and hæmorrhage into various

other viscera it must be considered to be allied to purpura or hæmorrhagic smallpox. I am aware that some of such cases have been said to be due to capillary embolism; but failing any large amount of evidence in favour of such an hypothesis, it seems at least as likely that the hæmorrhage may be due to some altered condition of the blood or of the tissues which hold it within bounds.

The two cases detailed above, however, are different. They are local extravasations of greater or less extent, one of which was associated with evidence of embolism in other viscera. The local changes in the damaged parts were also not unlike those found in hæmorrhagic infarcta; and though it is true that no plugs were found in any of the vessels of the heart, this is so often the case in other instances of undoubted embolism in various viscera that it can hardly be considered to disprove their occurrence at some earlier period in these cases.

In both cases death occurred somewhat suddenly, but though they had other and severe diseases, which in one case certainly, and in the other very possibly, would have terminated fatally before long, yet the state of the muscular tissue of the heart is not without interest as some explanation of the immediate cause of death.

December 21*st*, 1875.

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12. *Aneurysm of the septum of the heart; phthisis; contracted kidneys; general extreme atheroma, with aneurysm of the left internal iliac artery, and thrombosis of the vena cava inferior and both iliac veins.*

By J. Wickham Legg, M.D.

William H—, sixty years of age, was admitted on September 27th, 1865, into St. Bartholomew's Hospital under the care of Dr. Black. For the following clinical notes I am indebted to Dr. Bridges, the house physician.

The patient, an old man, with considerable ascites and some anasarca of feet, is a plumber and painter by trade. He is very thin and wasted, has a sallow complexion and thin hair. There is no blue line on the gums.

In the beginning of the year he had "rheumatism" and swelled legs, and for the first time a cough came on in the summer; but at the same time he was fairly well until about three months before admission, when he slowly lost strength and appetite, and suffered from constipation.

Physical examination.—Motions contain bile, and are apparently healthy. Urine is cloudy from urates, but contains no albumen, sugar, nor indican. The feet and ankles are anasarcous. There is considerable ascites and anasarca of the abdominal walls. The liver dulness reaches as high as the lower border of the fifth rib. No sign of enlargement below. Percussion over chest normal, save bases behind, which are absolutely dull, and where no respiratory murmur may be heard. The apex of heart beats in the nipple line, and much below it. Sounds are normal but intermittent. A loud diastolic (not præsystolic) murmur is heard occasionally at apex. When the heart's sounds intermit, the next beat does not carry the murmur. Pulse 34, small and weak. Arteries tortuous.

October 6th.—Both ascites and anasarca seem to be increasing, the latter invading the legs, and becoming extreme.

21st.—Friction-sound heard all over right lung.

November 1st.—He began to grow weaker.

5th.—He was delirious, gradually losing his senses and dying exhausted on November 8th.

Examination twenty-four hours after death.—Chest and arms very thin. Belly much swollen. Legs œdematous, particularly the right.

The friends forbade the head to be opened.

Both pleuræ adherent at apices. The pleuræ greatly puckered and opaque in places corresponding to hardness scattered through the lungs. These hard places being cut into, show in nearly all cheesy spots, from peas to nuts in size, surrounded by yellowish, round, solid bodies of the size of mustard seeds. These occur in groups, and at the apex are surrounded by highly pigmented lung tissue. No miliary tubercles seen. The bases of both lungs emphysematous.

The pericardium natural; the heart of natural size; the coronary arteries dilated, atheromatous, and tortuous. The heart muscle is of a brown red colour. There are no clots in the heart. The tricuspid valve is much thickened at its edges and opaque. The left side shows no signs of an acute endocarditis, past or present. The

aortic and mitral valves are distinctly atheromatous, the aortic sigmoids having calcareous plates at their attachment to the aorta, and are much stiffened. At the base of the left ventricle, between two aortic valves, where the large flap of the mitral is attached, in fact, in the undefended space, is a pouch which would take about half a small marble. It approaches a hemisphere in shape, the mouth being the widest part and nearly circular, the diameter varying from 17 to 20 millimeters. The walls of the pouch are formed of a thin semitransparent tissue, and are crossed by bands of membrane arranged like the musculi pectinati of the right auricle. The convex surface of the pouch, on the right side of the heart, is formed by the attached part of the tricuspid valve and part of the wall of the auricle adjoining. The pouch is not perforated, and there is no roughening or opacity of the endocardium of the left side around the pouch.

The peritonæum holds a very great quantity of a dark but clear fluid. The bowels are not adherent to each other; there are no false membranes. The peritonæum seems opaque and macerated looking, but it is not thickened. In many places, especially over the cæcum, it is highly pigmented in small patches. There are also numerous ecchymoses, and the larger of the small vessels seem full of blood. Over the liver is a thin layer of blood in substance, and between the coils of the bowels are some small clots. There is a particularly large ecchymosis, the size of half a crown, over the meso-colon of the ascending colon. The mucous membrane of the stomach and intestines quite sound.

The spleen very small and wasted.

The portal vein contains only fluid blood. The gall-ducts natural. The liver, though small, looks quite natural in appearance.

The supra-renal bodies natural. The kidneys small, the capsule tearing off in layers or having a rough surface, in many places cystic. On section the kidney is red, having a cortex wasted to a few millimeters in breadth, very confused in structure, and tough. Bladder empty.

The aorta much dilated; there is extreme atheroma, so that an elastic shining piece or one not thickened cannot be found. The same may be said of the iliac arteries, common, internal, and external. There is an aneurysm the size of a walnut on the left internal iliac artery. This aneurysm presses upon the accompanying vein, and at that spot begins a thrombus, adherent to the back part

of the vein, grey in colour, but not filling the vessel. This extends up into the inferior vena cava, the mouth of the iliac being covered by a large thrombus as big as a walnut, adherent to the vein about the opening of the iliac, but not elsewhere. It sends a prolongation down into the right iliac vein, which passes through the common iliac into the internal and external iliacs, but not into the femoral vein. In the right internal and external iliac veins the thrombus is adherent to the walls, filling the vessels, and of a grey colour.

This case seems to show that aneurysms of the septum may arise without foregoing endocarditis, and that Pelvet has thus gone too far in asserting this as the sole cause.[1] In this case it seems to me most likely that some imperfect development of the heart was the cause of the aneurysm, and we have like cases on record in our 'Transactions,' where it would seem that the aneurysm owned a congenital origin.[2] In this case there is certainly no evidence of an acute endocarditis, past or present.

I wish to draw especial attention to the following extract from Thurnam's Essay on Aneurysms of the Heart:—"It is well known to anatomists that the highest point of the septum which occupies the angle between the posterior and right aortic valves, and which, in some instances of congenital malformation, is deficient, is in the human subject formed not of muscular fibres but simply of the endocardium of the right and left ventricles, almost in apposition and strengthened only by the interposition of a little fibrous tissue continuous with that of the aorta. In many ruminant animals this point is well secured by an osseous plate; but in man as a comparatively weak spot it is perhaps probable that occasionally it may become the seat of aneurysmal dilatation. I am not, however, in possession of any fact which proves this to have occurred."[3]

This passage is noteworthy, since it not only shows the great pathological foresight of the writer, but likewise effectually disposes of the claims of later continental authors[4] to have been the first to describe the undefended space. Indeed, if any one after Thurnam may lay claim to the merit of this discovery, the name of Dr. Peacock might be brought forward, as in 1846, in a commu-

[1] Pelvet, 'Des Anévrysmes du Cœur.' Paris, 1867, p. 52.

[2] Peacock, 'Trans.' of this Society, 1846, vol. i, p. 61. Hare, ibid., 1865, vol. xvi, p. 80.

[3] Thurnam, 'Med. Chir. Trans.,' 1838, vol. xxi, p. 222.

[4] Hanska, 'Canstatt's Jahresbericht, f. 1855,' Bd. I, p. 66.

nication of his to our Society, the "undefended space" is spoken of,[1] a name very appropriate, and on which it would be hard to improve. It is a pity that this undefended space should receive so little attention in the elementary works on anatomy.

January 4th, 1876.

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13. *Aneurysms of the mitral valve.*

By J. Wickham Legg, M.D.

John M—, aged 37, was admitted into St. Bartholomew's on August 11th, 1875, under the care of Dr. Church.

For the clinical notes I am indebted to Mr. Macready and Mr. Bott, the house physicians.

He is a cheesemonger by trade, and has not been a drinker. Five weeks before admission, while coming home from Kilburn, he was very sick. The feet were then swollen. Feet and legs now very œdematous, and belly swollen. Pulse 82, fair volume, slow. Urine faintly smoky, sp. gr. 1018, highly albuminous. Under the microscope, numerous hyaline, medium-sized casts, granular in part, showing also epithelium cells far advanced in fatty degeneration. Many free epithelium cells also far advanced in fatty degeneration. Blood-corpuscles are scanty. At the heart there is a loud systolic mitral murmur. During the month of September the urine remained very scanty, scarcely ever more than a pint in the twenty-four hours and highly albuminous, usually one half or three quarters of the volume of the urine being filled with the coagulated albumen, and sometimes three quarters. The anasarca was general. At the end of the month a double murmur developed itself at the apex of the heart. On October 7th the presence of sibilant and sonorous rhonchi over both sides of chest was noted; also dulness at both bases. The urine remained highly albuminous, but more in quantity, until the man died, with an increase of bronchial symptoms on November 11th, 1875.

Examination forty-five hours after death.—Belly and legs much swollen.

[1] Peacock, loc. cit.

The right pleura holds about two points of a clear fluid ; the left about three. The peritonæum holds nearly a gallon of a fluid which is clear when first escaping, but afterwards becomes mixed with shreds of lymph.

The left lung collapsed throughout save at apex. The right lung less so ; the lower lobe only is solid.

The pericardium is adherent to the heart at the back part near the apex for a space the size of a shilling, and there are also adhesions between the inferior and superior cavæ. There are two white patches on the front surface of the heart. The heart is much larger than natural ; apparently more enlarged on the left than the right side. The aortic valves let water through readily. There is nothing remarkable on the right side ; very slight dilatation of the tricuspid orifice. The appendix on the left side is free from clot. The endocardium of the auricle over the small flap of the mitral is studded with vegetations, none bigger than a mustard seed, for a space which might be covered by a florin. On the large flap of the mitral are seen two bulgings, one at the edge of the valve close to the apex, rather flattened and not bigger than a split pea ; the other close to the first and seeming to run into it, but it is placed more towards the centre of the valve and is of the size of half a hempseed or bigger. A chorda tendinea passing into the apex of the valve has been torn loose from its attachment to the musculus papillaris ; another, close to its attachment to the valve, shows a spindle-shaped enlargement the size of a hempseed ; it is hard and seems to be made up of lime salts. The edge of the mitral valve shows small granulations ; these are much more marked on the small flap. On the ventricular surface of the large flap are two openings leading into the substance of the valve. One is in the middle of the valve and corresponds to the smaller bulging on the auricular surface. The opening is surrounded by vegetations of the size of mustard seeds. A probe can be passed into the smaller bulging through the opening. The other opening is close to the edge of the valve, but is not surrounded by vegetations ; it leads into the larger cavity. The aortic valves are stiff, having many granulations along their edges ; the substance of the valves is opaque.

The liver small, soft, and flabby, taking the print of the fingers. On section the surface is coarsely marked ; few red streaks in comparison with the amount of yellow present.

Spleen large; it shows two infarcts, which are triangular and of pale straw colour.

The stomach holds a quantity of altered blood.

Kidneys of natural size; the surface somewhat granular, pale, studded with white points, and a few red ramifying vessels; several cysts and scars of numerous infarcts. On section the cortex is seen to be very pale, opaque yellow, narrowed and showing a great contrast to the pyramids which are of a pale purple. All trace of striation of the cortex lost.

This case is published on account of the rarity of valvular aneurysms, not because any new doctrine can be established. I only wish to express my belief that these aneurysms arise from an ulcerative endocarditis, which causes a weakening of the valve at the place of ulceration, and the weakened spot being unable to resist the impulse of the blood at each systole of the ventricle, becomes bulged in the direction opposite to that of the course of the circulation.

January 4th, 1876.

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14. *Congenital malformation of the aortic valves, consisting in the existence of two segments only.*

By W. S. Greenfield, M.D.

This case was one of an abnormal condition of the aortic valves, resulting from defective development, and consisted in the existence of only two instead of three semilunar segments.

The heart was removed from a man 68 years of age, who was admitted to St. Thomas's Hospital on December 28th, 1875, under the care of Mr. Mac Cormac, for severe accidental injuries, from the shock of which he died on December 31st.

Post-mortem, twenty-six hours after death.—Body of a strongly-built muscular man. (The conditions due to the injury need not be detailed.)

Lungs.—Right weighed 29 oz.; left 25 oz. Both emphysematous at the upper part, much congested posteriorly and at the base.

Liver weighed 67 oz., slightly fatty, otherwise normal.

Spleen weighed 9 oz., was large, somewhat pale and soft; on section, the pulp, greasy and soft, easily washed away.

Kidneys.—Right of normal size, weighing 6½ ounces, capsule somewhat adherent, organ of generally somewhat fatty appearance; cortex slightly wasted. Left, contained two large thin-walled cysts, measuring 3 and 4½ inches in diameter respectively, containing clear yellowish, watery fluid; there were some other smaller cysts, and the renal substance was much atrophied.

Brain weighed 49 oz. Extreme atheroma of both internal carotids, the right being nearly occluded. General atrophy of brain-substance, with some thickening and opacity of arachnoid.

Heart of large size, weighing, after removal of coagula, exactly 16 ounces. The right auricle distended by a mass of recent clot, and the right ventricle also contained a mass of coagulum, which extended into the pulmonary artery. The left side contained only a small quantity of clot. The enlargement of the heart was general, and did not appear to affect specially any particular part all the cavities being somewhat dilated and their walls thickened.

Right auricle, endocardium opaque, some dilatation. A well-marked fossa ovalis with a communication which readily admits a probe to the left auricle.

Right auriculo-ventricular orifice normal, measuring 14 centimètres (5½ inches) in circumference. Tricuspid valve normal. Right ventricle slightly dilated and its wall a little thicker than normal. Pulmonary valves normal in size and situation; slight atheroma of pulmonary artery at its bifurcation.

Left auricle somewhat dilated, but not notably so. Left auriculo-ventricular orifice measured 12·8 centimètres (5 inches) in circumference. Mitral valve had some slight thickening at its free border. Left ventricle slightly dilated, the cavity measuring 7 centimètres (2¾ inches) in length along its posterior wall. The wall of the ventricle measured from ½ to ⅝ inch in thickness at its thickest part near the septum, and the muscular tissue was extremely fatty and friable. aortic valves appeared to close well, but were not very carefully tested.

The aortic valve was found to consist of only two segments, situated respectively anteriorly and posteriorly. The segments were of nearly equal size, the posterior being apparently slightly the larger

of the two; they met well in the middle line, but the sinuses of Valsalva were considerably dilated, especially the posterior. The posterior segment was apparently normal, though considerably larger than natural, its free margin measuring $5\frac{1}{4}$ centimètres in width. There was some atheromatous thickening at the attached border, and the valve was generally somewhat thick and opaque. The anterior segment evidently represented the normal right and left anterior semilunar segments; it was of nearly equal size with the posterior and of similar shape, but nearly at its centre a narrow ridge or spur was found running from near its attached border on to the wall of the aorta. This ridge, however, did not extend to the free margin of the valve, which was uniform, and shewed no sign of a division. The sinus of Valsalva was considerably pouched, but only a very slight groove existed on each side of the ridge before mentioned. Both the right and the left coronary arteries sprang from this sinus, one near each extremity. The ridge which indicated the position of the adjacent edges of the normal right and left anterior segments occupied the usual situation, opposite the ridge between the right and left posterior segments of the pulmonary valve. There was some atheroma and calcification at the base of the anterior segment. The aorta was considerably dilated in the ascending part of the arch, measuring $14\frac{1}{4}$ centimètres ($5\frac{3}{4}$ inches) in circumference at one inch above the valves.

The specimen is one of a form of malformation which has been especially studied by Dr. Peacock, who has recorded instances in the 'Transactions' of the Society and elsewhere.

The condition, no doubt, originates during the course of development, probably by the growing together of the adjacent margins of the segments at a very early period. In this case the perfect adaptation of the valves to the closure of the orifice, and the accommodation of the posterior segment to the abnormal size and shape of the other segment would seem to indicate that the change was entirely developmental, and in no sense the result of inflammatory adhesion. A similar condition may, no doubt, originate in endocarditis occurring during infancy; but in all probability there is usually in such cases either stenosis or imperfect adaptation of the segments, or a growth of vegetations, which conditions give rise sooner or later to some symptoms. The entire absence of any history of cardiac affection in the present case, coupled with the fact that the general enlargement of the heart is fully accounted for by senile

changes and by the condition of the kidneys, goes far to prove that no ill effects resulted from the malformation. *January 4th*, 1876.

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15. *Stenosis of the tricuspid and mitral valves.*

By W. S. Greenfield, M.D.

Stenosis of the right auriculo-ventricular orifice or of the tricuspid valve is a condition of great rarity, so far as can be judged by the number of recorded cases, and its detection during life is usually considered almost if not quite impossible. Dr. Flint, in his work on 'Diseases of the Heart,' states that a tricuspid direct murmur is one of the rare curiosities of medical experience; and Niemeyer remarks that stenosis of the tricuspid is extraordinarily rare. The majority of recent writers on diseases of the heart scarcely mention it. Dr. Hayden[1] has collected fifteen recorded cases, three of which have come under his own observation. In the 'Transactions' of this Society only three cases are on record, one in vol. i (1848), by Mr. Quain; another in vol. ii (1850), by Mr. Pollock; and a third in vol. iii, (1852), by Mr. Pye Smith, since which no case has been brought forward. In the 'Transactions' of the Pathological Society of Dublin six cases are reported. According to Dr. Hayden, in all of the fifteen cases save one the stenosis of the tricuspid was associated with a similar condition of the mitral valve.

The present case offered some other features of interest in their bearing on the questions as to how far the condition of the heart may have been concerned in the fatal issue, and whether syphilis may have had anything to do with its production; it may, therefore, be narrated in some detail.

Caroline N—, æt. 43, married, no children, was admitted under the care of Dr. Stone, on November 5th, 1875. She says she has had very good health; never had rheumatic fever. She had two miscarriages nineteen years ago, and has suffered from "fits" for twenty years. No history of any symptoms of syphilis can be elicited on careful inquiry. Two months ago she had a slight

[1] 'On Diseases of the Heart,' Dublin, 1875.

attack of bronchitis, and some vomiting after food. She has lately had some œdema of the face and legs, and pain in the chest, but no cough. A fortnight ago she got out of bed at about 5.30 a.m., and after doing so fell on the floor; on attempting to rise she found that she had lost the use of the left arm. There was no loss of consciousness, and no convulsions, and she states that she could move the leg freely after the seizure. There was impairment of speech for three or four days; and a pain in the back of the head which came on at the time lasted for three or four days.

The patient came to the hospital to apply for admission, walked there, got an order for admission, went away, came again in the evening, and walked to her bed.

State on admission, November 5th.—There is no loss of consciousness or of sensation; speaks well. There is complete paralysis of the left arm, incomplete of the left leg; the tongue is directed to the left and the left upper lip droops, but there is no distortion of the mouth. Pupils equal. Pulse 96, very small and irregular; temperature, (evening), 98° F.

The heart is evidently enlarged, cardiac dulness increased in width; apex 5 inches from the middle line, in the fifth interspace. Action irregular; thrill at apex. A short rough systolic murmur is audible over the base; and a loud somewhat rough præsystolic murmur at the apex and just within it.

Urine, sp. gr. 1020, cloudy; faintly alkaline, no albumen.

November 7th.—Got up as usual this morning to have her bed made, and said she felt quite well. At 9.30 a.m., whilst in bed, she became suddenly convulsed, the right side being chiefly affected, and then comatose. On going to her the house physician, Mr. Rossiter (by whom these notes were taken), found her quite unconscious, breathing stertorously; face congested and livid; pupils contracted to the size of pinholes; conjunctiva insensible; foaming at the mouth. The head was turned to the right; no rigidity or twitching of limbs. Heart's action tumultuous, loud rhonchi all over the chest; pulse hardly perceptible. Mustard and linseed poultice was applied to nape of neck; calomel 5 grains; Ol. Crotonis ♏j, Enema Sennæ Co. The enema was returned, the croton oil was repeated three times without effect.

10.30 a.m. Looks rather better; face less congested. Still comatose.

2.30 p.m. Had another convulsive attack, is now livid and comatose.

Died at 8.55 p.m.; the coma increasing until death. No rigidity of limbs.

Post-mortem examination twelve hours after death.—Weather cold. Body somewhat wasted. No scars on legs. No nodes on tibiæ.

Pericardium normal.

Heart weighs 13 ounces. Looking at the organ from the front there appears a marked disproportion in the size of the right and left ventricles. The right, which is of large size and normal shape, projects considerably beyond the left at the apex; the left, which is very small, appearing only half the length of the right. The *right auricle* is of large size, and somewhat conical shape, the appendix distended. The *right auriculo-ventricular orifice* is greatly narrowed by the adhesion of the margins of the curtains of the valve, so as to produce a somewhat half-moon-shaped orifice, measuring 22 millimètres (nearly 1 inch) in length, and 13 millimètres (half an inch) in width. [It should be stated that these measurements were not made until after distension of the cavities, and therefore are larger than in the fresh state.] The margins of the orifice are free from thickening and vegetations, the edge is sharp, and shows no sign of having been produced by inflammatory adhesion. The chordæ tendineæ are normal. *Right auricle* somewhat dilated, its wall thicker than normal. *Left auricle* enormously dilated, of oblong shape in its upper two thirds, conical below; the wall greatly thickened, nearly 4 millimètres thick, endocardium thick and opaque. Appendix greatly distended, forming a separate cavity with a somewhat narrow orifice, and extending downwards in front, so as to overlap the upper part of the ventricle. *Left auriculo-ventricular orifice* greatly narrowed by the adhesion of the curtains of the mitral valve, leaving only a rigid ovalish aperture, into which only the tip of the forefinger can be passed, and which measures only 18 by 7 millimètres in width. This orifice does not, however, lead directly into the ventricle, but into a funnel-shaped space formed by the coalescence of the chordæ tendineæ and the curtains, which are also adherent to the wall of the ventricle, a narrower orifice opening towards the wall of the ventricle at its lower part. Seen from below, the chordæ tendineæ and valve curtains appear fused into a solid semicartilaginous mass. The cavity of the ventricle is very small, and its walls thin. Aortic orifice normal, though apparently small, valves free from disease.

Lungs.—R. 15 oz., L. 14 oz., both somewhat emphysematous. Bronchi deeply congested, with signs of chronic inflammation. Pos-

terior parts of both lungs of somewhat red colour, exuding bloody fluid on squeezing; but for the most part normal, and free from signs of old or recent congestion.

Pleura.—Right, normal. Left, with general, moderately tough cellular adhesions.

Liver.—Weight 2 lb. 14½ oz. Some adhesions of capsule to diaphragm. Over the surface of the liver, especially on the upper surface of the right lobe, are numerous deeply puckered and depressed cicatrices, typically syphilitic. Others also over the under surface of both lobes.

Spleen.—Weight, 7½ oz. Firm, tough, of deep red colour, as from chronic congestion, free from infarcts.

Kidneys.—Weight, 7 oz., small, capsule adherent, cortex wasted, very tough, surface somewhat granular, with some depressed cicatrices, apparently due to old infarcts.

Brain.—Dura mater opaque and thickened over vertex, sinuses normal. Superficial veins nearly empty, pia mater generally pale.

Vessels at base.—Both internal carotids somewhat rigid at their termination, left posterior communicating artery of remarkably small size. Internal carotid, middle cerebral, anterior cerebral, and posterior communicating arteries free from thrombosis. The basilar artery is filled by a firm dark thrombus in its anterior two thirds, the clot being apparently of quite recent origin, although formed before death. This clot extends into the left posterior cerebral artery, but only into that portion posterior to the junction of the posterior communicating artery. The left superior cerebellar artery, which is represented by two branches arising from the posterior cerebral close to the basilar, is completely blocked by coagulum. The outer two-fifths of the upper surface of the left hemisphere of the cerebellum is of dark mulberry-red colour, somewhat softened, and surrounded by a narrow zone of whiter colour.

Sections of the hemispheres show nothing abnormal above the level of the corpus callosum. Over the anterior and outer part of the roof of the right ventricle is a slight induration of the white substance. The anterior two thirds of the nucleus caudatus, with the anterior and upper part of the optic thalamus, are softened, of yellow colour and broken down; the right ventricle contains yellowish, slightly turbid fluid.

The condition of the other organs presents nothing abnormal.

Remarks.—The entire absence of symptoms pointing to the ex-

istence of such extensive disease of the heart-valves, which must have existed for a long period, is a point of interest. Moreover, the other viscera showed but scanty evidence of the influence of cardiac embarrassment. Stenosis of the mitral valve, to an advanced degree, seems to be often compatible with the enjoyment of good health, to judge from the frequency with which it is found, *post-mortem*, in the bodies of those who have not been suspected to suffer from heart disease. Nor does there seem to be anything in a moderate degree of tricuspid stenosis which could in any way increase the tendency to the production of symptoms. Indeed, it might be maintained with some reason that its existence, by equalising the circulation in the ventricles, although it might impede the general circulation, would tend to prevent engorgement of the lungs, and might therefore even have a beneficial influence on the general course of the disease.

The cause and the date of the disease seem very doubtful. The tricuspid stenosis had much the appearance of a congenital condition; but, from a comparison with other cases, I am inclined to suggest the possibility that the syphilis, which existed at some period, as is shown by the condition of the liver, may have been the cause.

The condition of the tricuspid valve was not diagnosed during life. The rough præsystolic mitral murmur may have been blended with the tricuspid murmur, but this I do not think likely, as the murmur was very localised. But it must be borne in mind that, the right ventricle being much enlarged and completely covering the left, the murmur heard may have been a tricuspid direct murmur. I am inclined to believe that the systolic murmur at the base was an auricular appendix friction murmur, as no other condition was found which could account for it. But is it possible that a tricuspid direct murmur might be heard in that situation? Had the patient been under observation for a longer period, and when in a better state of health, these points might have been ascertained. As to the possibility of a tricuspid direct murmur being distinguished from a mitral direct, where both are present, which has been denied, it need only be said that the diagnosis has been made by Dr. Gardner.

There remains the question whether the thrombosis of the cerebral arteries was due to the embarrassed action of the heart. I am rather disposed to believe that it depended on disease of the vessel of syphilitic origin, as the latter is so often the cause of multiple thromboses.

The rapidly fatal issue was probably due to the cutting off of the supply of blood to the pons by the plugging of the basilar artery, and the extreme contraction of the pupils is, from this point of view, a fact to be noticed. *January 18th*, 1876.

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16. *Occlusion of superior vena cava.*

By T. Henry Green, M.D., for A. W. Stocks.

Thomas D. G—, æt. 44, a short thick-set man, joiner, has had no particular disease except a carbuncle at the back of his neck in 1874. The capillaries of his face have been dilated all his life, but more markedly so since August, 1875. He has had since boyhood a more or less livid look, the other members of his family being rather pallid. He worked at his trade until the latter end of August, 1875, when it was noticed that when he slept he always made a loud roaring noise during respiration, perfectly audible in the room below his bedroom.

He came under my notice in September, 1875. He has a short thick neck, countenance heavy and suffused. His lips, ears, nose, and hands show a good deal of livid congestion. His breathing is difficult, and voice peculiar, giving one the impression that he has a bronchocele.

On removing his clothes, his neck, though usually thick, was found to be increased in circumference at the base by two inches. The posterior triangles had an appearance similar to that produced by emphysema, but they were not resonant. They did not pit on pressure, nor give to the touch any impression of enlarged glands. The chest above the level of the diaphragm, especially in parts, was studded with numerous clusters of dilated capillaries, the largest collection being over the cartilages of the left false ribs. The whole of the body above this level was œdematous and brawny, the left arm so much as to make the skin erythematous. The lower part of his body became œdematous only on the approach of death.

Not being able to lie down he sat in a chair with his chin on his chest, and always seemed to be in a state of semi-stupor, from which, however, he was easily aroused, but which gradually increased in intensity.

The upper part of his right chest was dull anteriorly, the posterior part entirely dull; vocal resonance was very much diminished. The left pulmonic sounds were fairly healthy; cardiac sounds natural; pulse normal. His condition continued much the same until his death, which took place on November 11th, except that the swelling in the neck had very distinctly diminished.

Inspectio cadaveris—twenty-six hours after death.

The dilated veins on the front of the chest have nearly disappeared. The right pleural cavity is distended with fluid, having flakes of lympth floating in it abundantly. The lung is pressed upwards towards the spinal column. The pleura is coated with a thick layer of lymph; on the front of the pericardium is a collection of jelly-like lymph; there is also about three ounces of fluid in the pericardium. The heart is natural except that the finger passed upwards through the right auriculo-ventricular opening is arrested in a *cul-de-sac*, the opening into that cavity from the superior vena cava being completely closed. The right auricle is conical in shape, with its apex as the root of the superior vena cava. The vena cava itself, for about $1\frac{1}{2}$ inches of its course from the auricle, is completely impervious, further from the heart its diameter is about $\frac{1}{6}$ of an inch, and its walls are apparently thickened.

Encircling this vessel, and encroaching somewhat on the other vessels springing from the heart, is a dense mass which is apparently the cause of the occlusion of the vein. Microscopically it showed very numerous cells of multiform appearance, spindle-shaped, many-tailed, &c., containing large nuclei.

There is no history of hereditary cancer in this case, and I am inclined to believe that the primary disease, namely, the thickening about the root of the vena cava, was of old standing, though in all probability not congenital, and that his death was caused more immediately by pleuritis resulting in effusion in the right chest and deposit of lymph in the mediastinum. Death was hastened no doubt by dyspnœa arising from pressure on the trachea and bronchi by the tumour. The interruption to the circulation had been largely compensated for by a collateral circulation, evidences of which were plainly visible on the chest during life. There was no dilatation of the external epigastric veins, the shorter course through the phrenic veins being taken to reach the inferior cava.

It is somewhat remarkable that within a very short time of the occurrence of the above case I met with another, having appear-

ances during life almost exactly similar to those recorded above, but in which was found after death a tumour about the size of the ungual phalanx of the middle finger projecting into the right auricle, and interrupting the flow of blood into the ventricle, the superior vena cava being free. *April 4th*, 1876.

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17. *Persistence of left vena cava superior, with absence of right.*

By W. S. Greenfield, M.D.

The specimen exhibited affords an instance of an abnormal mode of development of the great venous trunks, consisting in the persistence of the left instead of the right duct of Cuvier as the main channel for the conveyance of the venous blood from the head and upper limbs to the right auricle. It will be remembered that early in fœtal life the blood from the right and left brachiocephalic trunks respectively is separately conveyed to the right auricle by the right and left "ducts of Cuvier;" subsequently a transverse branch is formed, connecting the left brachiocephalic with the right, and the right duct of Cuvier forms the normal vena cava superior in man. The left is partially obliterated, its lower portion persisting as the coronary sinus, whilst the upper forms the oblique vein, the intermediate portion producing the "vestigial fold" of Marshall. Minor details may be omitted, and the reader referred to Mr. Marshall's memoir for a full account of the mode of development.[1] In some cases both ducts of Cuvier persist, and a "double" vena cava superior is thus formed, the blood from each brachiocephalic trunk entering the heart separately, or in some cases a larger or smaller transverse trunk connecting the two.

In the present case the left trunk alone appears to have persisted, the right opening into it; the right, if persistent, which is doubtful, being represented by only a very small vein: the usual condition being thus reversed, and the whole of the blood from the upper extremities and head entering the coronary sinus.

[1] "On the Development of the Great Anterior Veins in Man and Mammalia," by John Marshall, F.R.S. 'Philosophical Transactions,' Part I, 1850.

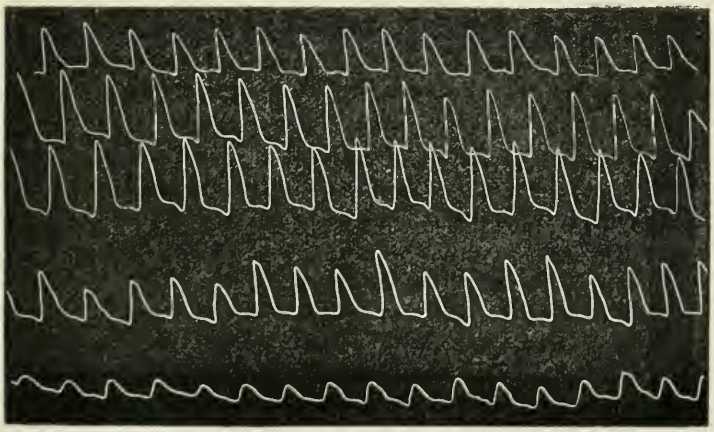
How far this condition influenced the course of the symptoms must be judged from the clinical history, for which and for the opportunity of bringing the case before the notice of the Society I am indebted to Dr. Stone, under whose care the patient was.

J. J. M—, æt. 39, a butcher; admitted January 5th, 1876; died January 23rd. He stated that he had been subject to slight winter cough for some time, but had been really ill for only eight weeks, at which period he was suddenly seized with cough, and had since that time occasionally spat streaks of blood on coughing. He had been very short of breath since his attack, and for six weeks his feet had been swollen. Had never suffered from rheumatic fever or other serious illness. He had been in the habit of drinking pretty freely beer, rum, and whisky.

On admission there was extreme dyspnœa, face livid, expression anxious, breathing rapid, inability to lie on the left side. Legs very œdematous, bedsores over sacrum and heels. Loud crepitation and bronchitic râles all over the chest. Heart-sounds feeble, no murmur. Tongue clean, bowels regular. Urine scanty, free from albumen.

January 10th.—General condition much improved. On examining the heart it appears to be hypertrophied, but overlapped by the emphysematous lungs. A somewhat doubtful systolic murmur is heard over the base of the heart. Pulse very regurgitant.

Woodcut 2.



No further notes are to be found, but the dyspnœa and other

symptoms gradually increased, the patient ultimately dying on January 23rd from erysipelas and gangrene induced by acupuncture for the dropsy of the legs.

The tracings of the pulse (*vide* Woodcut 2), and the other symptoms, led Dr. Stone to the belief that there was probably some obstruction of one of the large venous trunks.

Post-mortem.—Body stout; great œdema of both lower limbs, with thickening of the skin as from chronic congestion; gangrene of left leg as high as the knee, and inflammatory œdema over front of left thigh. The peritoneum contains about 8 pints of clear serous fluid, the right pleura $2\frac{1}{2}$ pints, and the left $1\frac{1}{2}$ pints.

The *pericardium* contains about $1\frac{1}{2}$ oz. of turbid serum, and there are adhesions over a great part of its anterior surface by recent lymph.

Heart large, weighing $22\frac{1}{2}$ oz.; circumference at base of ventricles 13 inches; the right semi-circumference 7 inches, the left 6 inches; left ventricle 5 inches in length, large, with rounded apex.

The right auricle small as to its body, but the appendix much distended.

The heart was unfortunately removed before the relation of the great vessels was traced, the following description being the result of such investigation as was possible afterwards.

The inferior cava opened in the usual situation, but further forwards and at a somewhat higher level than usual. The orifice for superior cava was completely absent, and the corresponding elongation upwards of the body of the auricle did not exist, so that the auricle appeared flattened and truncated at its upper part, the lower part and appendix being correspondingly increased in size. A small orifice, the size of a crowquill, was found close to but to the outer side of the orifice of the vena cava inferior, but no other external aperture could be discovered, and the relations of this opening could not be traced. On opening the auricle in front an orifice of large size was seen in the position of the entrance of the coronary sinus, which was situated just posterior to and below the opening of the vena cava inferior, a small and imperfectly developed Thebesian valve existing at its lower part. This enormously dilated coronary sinus and vein, passing round in the usual situation, was continued upwards by a large venous trunk lying in front of the pulmonary artery to the vessel formed by the junction of the left jugular and subclavian veins; the right jugular and subclavian, uniting in the

usual manner, passed obliquely to the left, joining the left brachio-cephalic trunk.

A small vein opened into the right brachiocephalic near its lower part, but its exact relations could not be determined. From its position it seemed probable that it represented the vena azygos major, but the azygos and superior intercostal veins could not be traced.

Woodcuts 3 & 4.

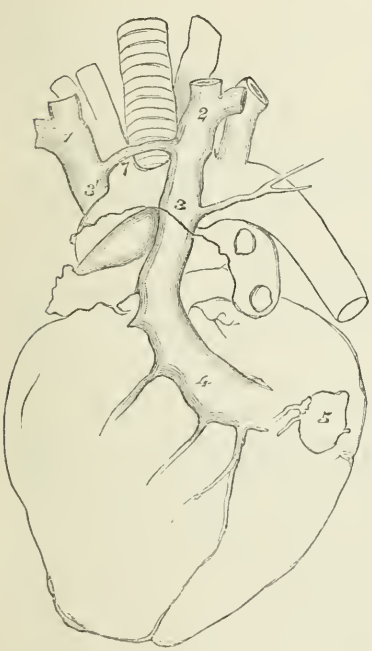


Fig. 1.

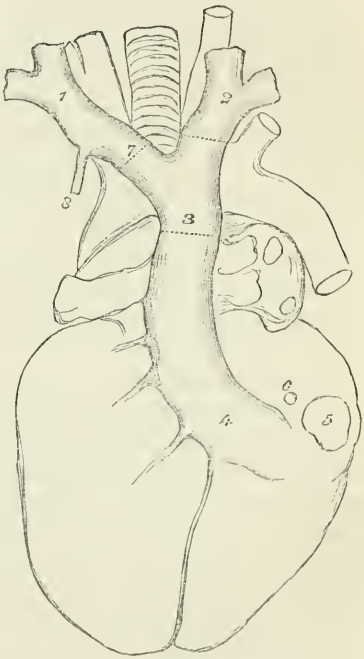


Fig. 2.

Semi-diagrammatic sketch to show relations of great vessels. Fig. 1, taken from Mr. Marshall's paper, to show persistence of *both* ducts of Cuvier. Fig. 2, from case of J. J. M—, showing persistence of *left* only. (About one third of natural size.)

1, Right, 2, Left brachio-cephalic vein. 3, Left, 3′, Right vena cava superior. 4. Coronary sinus. 5. Opening of vena cava inferior. 6. Small orifice, the relations of which were doubtful (? azygos vein). 7. Transverse communicating branch.

The heart is viewed from the left and posterior aspect.

The woodcuts are intended to represent, in a somewhat diagrammatic manner, the condition of the vessels (*vide* Woodcuts 3 and 4),

fig. 1 being taken from Mr. Marshall's paper, to show the persistence of both anterior primitive veins, for the sake of comparison. The dotted lines in fig. 2 indicate the point at which the vessels were accidentally cut before examination.

The veins of the neck were enormously distended with fluid blood. Right ventricle large, its walls thick. Left auricle large, funnel-shaped. Endocardium opaque. Mitral valve normal. Left ventricle dilated and hypertrophied. Aortic valves close imperfectly under a stream of water. Aorta extremely atheromatous and rigid throughout, the inner surface of the vessel resembling the bark of an oak tree.

Lungs extremely emphysematous, bronchi with signs of chronic congestion. Liver normal. Spleen somewhat congested. Kidneys large, firm, with very distinct and prominent Malpighian tufts; capsule slightly adherent.

Nothing else abnormal of note in the viscera.

Taking into consideration the other morbid conditions, it seems to me very questionable whether the abnormal condition of the vena cava had any influence in the production or on the course of the symptoms.

In two ways only can it be supposed that venous obstruction may have resulted in some degree. The passage of the blood along the coronary vein and sinus was necessarily less direct than usual, and from the fact that it must pass round the back of the heart, it is not improbable that when the heart was enlarged some compression might be exerted upon the vessel against the spine. Possibly, too, the direction of the blood-current in the auricle might prejudicially affect the return from the inferior cava. But I should be unwilling to ascribe much importance to either of these factors. *April 4th*, 1876.

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18. *A case of acute thrombosis of the superior mesenteric and portal veins, attended with rapidly fatal collapse.*

By C. Hilton Fagge, M.D.

On January 26th, 1875, I was asked to see, with Dr. Finch, of Blackheath, a lady, æt. 34, who had that morning been seized with acute abdominal pain and collapse.

She was a person of pale and sallow complexion. She had in childhood had an abscess in the lumbar region on one side, and a slight scar remained from this. She had been attended by Dr. Finch for many years, and had often complained of pain in the back; she had also been subject to bilious attacks. She was married in the early part of 1874, and on December 22nd she was confined with a healthy female child. The labour was natural, although protracted. Her recovery was favorable, but on January 6th she felt pain in the left leg, and phlegmasia dolens showed itself on the following day. The affection appeared to be confined to the calf; there was neither pain nor tenderness in the groin. The pain and swelling of the limb subsided in about a week, but the right leg then became affected in a similar way. She was kept in a state of perfect rest, and the affected parts were wrapped in cotton wool. On January 11th she had a troublesome diarrhœa; it may be noted that after her confinement she had had one or two bilious attacks, such as she had been subject to before her marriage. By January 25th the condition of the right leg had improved so much that Dr. Finch proposed to discontinue his daily visits.

On the 26th she was suddenly seized at 6 a.m. with violent pain in the abdomen, and with vomiting. Her husband fetched Dr. Finch to her at 9.30. She was then found to be in a state of absolute collapse, and apparently dying; her eyes sunken; her pulse almost imperceptible; pain coming on in paroxysms, but never completely intermitting; retching severe; and frequent vomiting of a rather viscid blood-stained liquid in small quantity. Dr. Finch said afterwards that the agony seemed to him more severe than any which he had ever seen a patient undergo. She was perfectly conscious.

I saw her with Dr. Finch and with Mr. Jeken, of Eltham, at about 2 p.m. The pain had then passed off to a great extent, and the pulse had somewhat rallied. She was dozing, and seemed to be hardly conscious. The abdomen was perfectly flaccid and soft. There was no tenderness anywhere, nor any fulness; no indication of any local mischief could be detected on manipulation. I could not form the slightest idea as to the cause of this sudden illness. The pain soon returned, and she died at about 5 p.m.

A *post-mortem* examination was held the following day, at which Dr. Finch, Mr. Jeken, and I were present.

The body was fairly well nourished.

When the abdomen was opened, the first thing that attracted

observation was the extreme congestion of part of the small intestine. This was of an intense purple-black colour. It contrasted in a very marked way with the rest of the bowel, which was even more pale than usual. The congested part was limited, both above and below, by a well-defined edge. It began, above, about four inches from the termination of the duodenum; and below it ended at about the middle of the small intestine. The congestion extended a little way, but not far, into the mesentery. The surface of the affected part was smooth; and there was no peritonitis—unless a small shred of lymph, at one spot, was proof of peritonitis. The intestine felt massive, and on section its coats appeared to be œdematous. Its internal surface was deeply reddened, and covered with shreds of mucus, so that at first it seemed as though the mucous membrane itself was being detached. It contained a considerable quantity of a thin reddish fluid, very like what had been vomited during life.

This congested state of the jejunum was so like what would have resulted from internal strangulation of the bowel, that I searched very carefully for any hernia or other cause of obstruction. But none could be found. Moreover, the affected part of the intestine was by no means distended; it was not larger than the rest.

My first impression on finding the jejunum affected in this remarkable way was that it must be due to embolism of the superior mesenteric artery, secondary to puerperal endocarditis affecting the valves of the left side of the heart; but on examining that organ I found that all its valves were perfectly healthy.

The real explanation of the state of the intestine, however, at once became apparent when we cut through the mesentery to remove it. The branches of vein coming from the affected part were then found to be distended with adherent coagulum. This condition was traced up the superior mesenteric vein into the trunk of the vena portæ, nearly to the point where this vessel begins to break up again into branches. The thrombosis extended into tributaries of the superior mesenteric vein, which lay somewhat beyond the area of the congested part of the intestine.

Towards the upper extremity of the thrombus it was softening, and adherent to the wall of the vein. But within the mesentery itself the rootlets of the vein were distended to an extreme degree by clot which was there perfectly solid, and evidently of more recent formation. I may add that after having been kept in spirit for some

length of time the plugged veins could be felt through the serous membrane, to within an inch or two of the bowel, as hard round cords.

The rest of the abdominal viscera were apparently healthy. The stomach was pale, but rather distended. The liver was pale, and seemed to be fatty. The spleen was of natural size, and of tolerably firm consistence.

The femoral veins were each of them plugged with softening adherent thrombi. The same condition extended up the iliac veins into the vena cava, and along this as far as the entrance of the renal veins, above which point the cava was patent. The walls of the plugged veins seemed to be somewhat thicker than natural; and they were also markedly blackened. These appearances were no doubt of recent origin; but the vena cava had by no means been in a normal condition before the thrombosis began. On the contrary, it was flattened, and narrow, and imbedded in a quantity of very firm fibrous tissue, which also enclosed the aorta and the trunks of the sympathetic nerves, and bound them all tightly down to the spine. So marked was this change that I had some difficulty in discovering the vena cava and aorta in the midst of the hard substance which surrounded them; and on afterwards attempting to dissect off the vena cava from the spine I found that it adhered so closely that I left portions of it behind.

The induration of the connective tissue in front of the lower lumbar vertebræ extended downwards along the brim of the pelvis, involving the lower branches of the lumbar plexus. It extended round the sides of the lumbar vertebræ. These vertebræ themselves did not seem to have been affected with any definite disease. The inter-vertebral discs were all healthy; and the interior of the bodies of the vertebræ appeared perfectly normal. But the left side of the last lumbar vertebræ seemed to be slightly excavated; and on scraping it with the knife I found that it felt rather rough, and that I could cut away some small portions of osseous substance, embedded in the tough fibrous tissue.

Remarks.—This case presents several points of interest. The induration of the connective tissue in front of the lumbar vertebræ, around the aorta and vena cava, is important as affording an explanation of the pain in the back to which the patient had from time to time been subject from childhood. Doubtless it is often the case that pain which returns in the same spot from year to year, is thus

due to the residues of former active mischief, involving nerves in cicatricial tissues, or otherwise interfering with them.

But the special value of the case is as an illustration of portal thrombosis—*pylephlebitis adhesiva*—a disease which (as I believe) very rarely is attended with most acute symptoms, or terminates rapidly in death.

One case somewhat similar, in so far that the occurrence of thrombosis of the portal vein was associated with the supervention of collapse, was observed by Dr. Wilks in the year 1858. A woman, æt. 32, was admitted into Guy's Hospital with jaundice and a tumour situated in connection with the lower edge of the liver. While there she was taken suddenly ill, and became collapsed, so that it was thought a cyst or one of the abdominal viscera might have given way; and in two days she died. There was no tympanitis or any evidence of peritonitis by which the suspicion of rupture or perforation might have been corroborated. On *post-mortem* examination the peritoneum was found to be healthy, as were also the stomach and intestines. The tumour was found to be a mass of cancer in the substance of the liver, involving the outer wall of the gall-bladder. The gall-bladder itself contained hundreds of calculi. The whole of the branches of the portal vein to their finest twigs were filled with coagulated blood, so that casts of them could be drawn out. These were becoming decolorised, and had evidently been there for two or three days. Microscopically they were found to contain nuclei, resembling those of the cancerous growth. Dr. Wilks goes on to ask the question, "Did the collapse three days before death arise from the sudden arrest of circulation throughout the liver?" The liver itself contained an excess of fat, but was otherwise healthy. The hepatic vein and its branches were quite empty. *April 4th*, 1876.

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19. *Double mitral valve.*

By W. S. Greenfield, M.D.

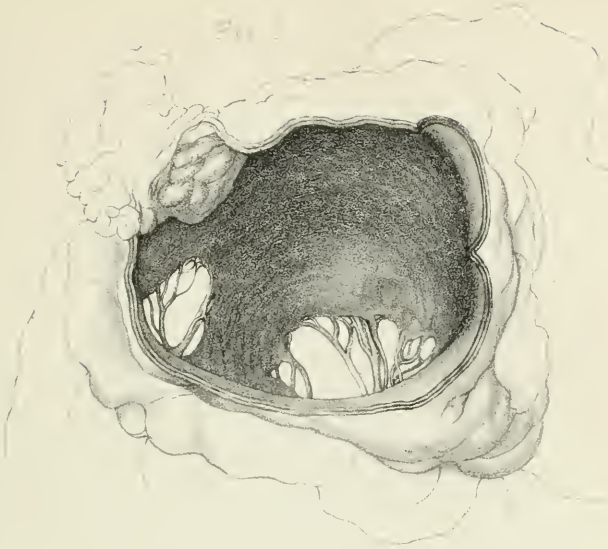
The specimen exhibited, which was removed from the body of a man 28 years of age, who was under the care of Dr. Bristowe, and died from the results of acute myelitis, is an example of a

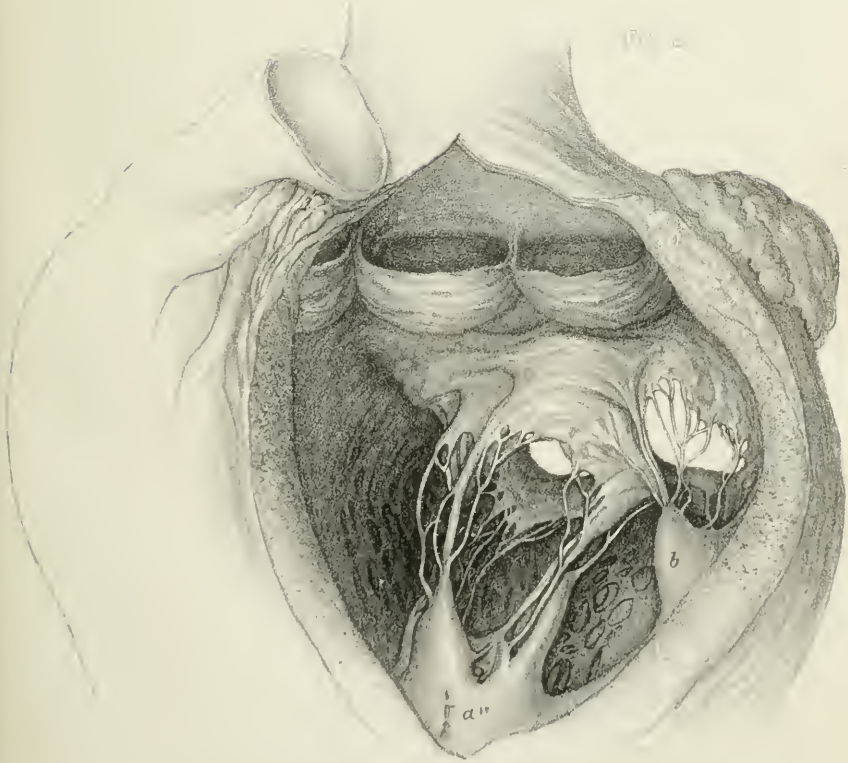
DESCRIPTION OF PLATE IV.

Plate IV illustrates Dr. Greenfield's case of Double Mitral Valve. (Page 128.) From drawings by Mr. G. Burgess.

FIG. 1. Seen from the auricle.

2. This shows the cavity of the left ventricle laid open in front by an incision near the septum. The larger valve is seen receiving chordæ tendineæ from posterior musculi papillaris *a*, the smaller from anterior *b*. (The smaller orifice appears much larger than in the fresh condition, the valve having been necessarily stretched in the process of preserving the specimen.)




b
a''

curious malformation of which I can find no other instance on record. There was no evidence either in the clinical history to the condition of the organs after death that the malformation gave rise to any pathological results.

The heart was of somewhat, but not notably large size; externally its appearance was normal, and the valves, on testing with a stream of water, closed perfectly. But on looking at the mitral valve on the auricular aspect, two distinct orifices, of unequal size, were seen to exist. Whilst at rest the smaller orifice appeared as if it were an irregular opening in one of the curtains, produced by rupture, but on passing a stream of water into the ventricle this smaller orifice as well as the larger was found to close perfectly by small curtains. The smaller orifice was about half an inch in diameter, and of rounded shape. Whilst in action this valve came up to the same level as the other, during diastole falling back against the wall of the ventricle.

On inspection from the ventricular aspect, it became evident that the mitral valve was divided into two unequal halves, apparently by developmental adhesion between the two curtains at one point, each of the two portions being provided with distinct chordæ tendineæ, and complete in itself. The smaller valve, situated anteriorly and to the left, received its chordæ tendineæ entirely from the small anterior musculus papillaris; whilst the posterior and larger portion was entirely supplied by chordæ tendineæ from the large fleshy bundles on the posterior wall. There was an entire absence of thickening or other sign of old disease about the valves; the curtains were perfectly translucent and healthy looking. The upper surface of what must be called the septal portion was smooth, and sloped gently down on both sides, so that no obstruction to the flow of the blood current appeared to be produced by it. Moreover, there were none of the associated lesions of mitral stenosis.

I can suggest no other hypothesis of the mode of development of this malformation than that of an adhesion at a very early period of the growth of the organ.

The drawings show respectively the views of the valve from the auricular and ventricular aspects. (*Vide* Pl. IV, figs. 1 and 2.)

May 2nd, 1876.

20. *Aneurysm of the arteria innominata, and one of the aorta: the latter opening into the trachea.*

By Thos. B. Peacock, M.D.

This specimen was obtained from a male, æt. 36, formerly in the marines, but latterly a gardener, who was admitted into the Victoria Park Hospital on November 4th, 1875. He had served for only a short time in the marines, and was invalided from the service ten years before his admission into the hospital for an injury of the head, followed by erysipelas and abscess. After his recovery from that illness he enjoyed good health till three months before, when he had a bilious attack accompanied by jaundice, and took cold while under the influence of medicine. He then suffered from cough, expectoration, and shortness of breath, with pain in the chest; and these symptoms continued till the time of his admission into the hospital. He then had pain in the left side, extending across the chest to the right shoulder; his breathing was short, and he had a hollow voice and a husky abortive cough.

There was an obvious pulsation above and behind the upper part of the sternum, and a harsh systolic murmur was there heard, as also in the carotid arteries and to the left of the spine behind. There was increased dulness at the upper part of the sternum, and a double murmur at the base of the heart.

The left pulse was smaller than the right, and the right pulse had a decidedly regurgitant character. There was some stridor with the respiration, especially on the left side. On January 10th, 1876, there were slight streaks of blood in the expectoration, and soon after he brought up a large quantity of blood, and immediately died. An aneurismal dilatation, about the size of a hen's egg, occupied the whole of the arteria innominata and compressed the branches of the descending cava, and the trunk and recurrent branch of the right pneumo-gastric nerve. At the arch of the aorta, immediately to the left of the orifice of the arteria innominata there was a circular opening sufficiently large to admit the end of the little finger, which opened into a sac about the size of a chestnut. This had compressed the lower end of the trachea above its bifurcation, and had opened into that passage. The ascending and transverse portions of the aorta were dilated, and the coats of the

vessel were rough and very much thickened. The aortic valves were incompetent. The heart was somewhat large, weighing about twelve ounces, and the left ventricle was hypertrophied and dilated.

May 2nd, 1876.

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21. *Malformation of heart.*

Stenosis at the commencement of the conus arteriosus of the right ventricle and at the origin of the pulmonary artery; aperture in septum ventriculorum and aorta arising partly from right side; foramen ovale and ductus arteriosus closed. Cyanosis.

By Thos. B. Peacock, M.D.

E. S. R—, æt. 4, residing at Notting Hill, admitted into Victoria Ward, St. Thomas's Hospital, January 14th, 1876, labouring under cyanosis. She had previously been for about two months at the Children's Hospital, Great Ormond-street, under the care of Dr. Dickinson, to whom Dr. Peacock was indebted for having the case sent to him.

From the account given by the mother, it appeared that this child was the youngest of the family, there being two other children living, and one having died of scarlet fever. The child was born at the full period, and about six hours after birth, its heart was observed to beat violently, and its colour was always higher than that of the other children, but there was otherwise nothing peculiar to attract the mother's attention till it was twelve months old, when the breathing was observed to be short; when two and a half years old the lividity became marked, and about ten weeks before her admission into St. Thomas's, she spat some blood and was in consequence taken to the Children's Hospital.

Her father and mother are both alive; the former being thirty-one, the latter thirty-three years of age. Her father has had two attacks of rheumatic fever and has been subject to palpitation all his life. Her mother is a small delicate-looking woman and has lost her father and an aunt suddenly from heart disease and a sister by consumption.

When the child was admitted into the hospital, the symptoms of cyanosis were very marked. Her cheeks were of a deep purple

colour, the hands and feet were very livid, the ends of the fingers and toes clubbed, and the nails incurvated. She was very thin. The chest was very prominent in front, especially at the lower part and on the left side, and the area of dulness on percussion in the præcordial region was much larger than it should be. The child was then so irritable and timid that it was impossible to get a satisfactory examination of the chest, but the two sounds of the heart were distinctly audible and apparently without any murmur.

She continued much in the same state till March the 10th. During this time her temperature was generally slightly above the normal standard, ranging in the mornings between 99·3° and 98°, and in the evenings between 99° and 98°. Her breathing was at all times very hurried, and she had a troublesome cough, and occasionally spat blood in quantities varying from ʒij to ʒiss; and before each expectoration, she became excited and restless and her temperature rose, on one occasion attaining 100°. After the expectoration she became quieter and appeared to be relieved by it. She was always very livid, and sometimes her face and extremities became almost black. She became less timid and fractious and repeatedly allowed her chest to be examined, and on each occasion the heart sounds were distinctly heard and were unattended by any murmur and very much resembled the fœtal tic tac.

On the 10th of March she had a slight expectoration of blood, and coughed and sneezed, and as there had been some cases of measles in the ward, and one of the patients had occupied the bed next to her, it was thought that she was sickening with rubeola. The symptoms did not, however, more fully develop themselves, and she continued much in the same state, though her cough was more troublesome and she occasionally expectorated blood. On the 25th two or three slightly elevated livid spots appeared on the abdomen, and the following evening her temperature had risen to 99°, and on the 27th to 100·3°. On the 28th the morning temperature was 99·2°, and on the 29th the morning temperature was 99·2°, and the evening 102·4°. On the 30th the temperature was 102·5° in the morning, and 104·9° in the evening. On the 31st it was 104·8°, and a copious livid rash was found on the skin of the face and trunk, with large blotches in places. A slight whiff was heard at the base of the heart with the systole. She was now transferred to the ward appropriated to the cases of measles. On the morning of the 1st of April the eruption was very dark and blotchy, and she was much

prostrated. She breathed very hurriedly and coughed and spat some blood. The temperature was about 102°, and in the afternoon it rose to 103°, and at 10 o'clock at night it was 104·8°. She died at 4.45 on the following morning.

The *post-mortem* examination took place on April 3rd.

The height of the body was 3 feet 4 inches, and its weight 17 lbs.

The thymus gland was of large size.

The lungs were very much congested, as indeed were all the other organs of the body. The liver weighed 18¼ oz.; the spleen, 2½ oz.; the kidneys, 3½ oz.; the brain 38 oz.

The heart was of large size for the age of the subject, and weighed 3½ oz.

It was unduly wide, being 26 Paris lines transversely (58.5 mm. and 2·3 English inches), and 24 in the vertical direction (54 mm. and 2·13 English inches).

The right ventricle was greatly hypertrophied and the walls firm, and the cavity, when the organ was first removed from the body, was very small. The right auricle and ventricle and the pulmonary artery contained firmly coagulated but dark blood. The walls of the left ventricle were less firm.

The descending and ascending cavæ entered the right auricle as usual; the Eustachian valve was very distinct. The auricle was large and the foramen ovale entirely closed. The right ventricle consisted almost entirely of the sinus, the conus arteriosus being very small. The pulmonary artery arose as usual from the conus arteriosus of the right ventricle, but its orifice was small, when cut measuring only 13 lines in circumference (29·25 mm., 1·15 English inches); it was provided with only two valves, which were transparent and free from thickening, and readily closed the opening. The origin of the artery was connected with a small cavity, not more than half an inch in length, which represented the conus arteriosus, and this again was separated from the sinus by a second opening only admitting the passage of a blowpipe 7 Paris lines in circumference (15·75 mm., ·62 English inch). This opening was surrounded by a ring of very minute vegetations. There was an aperture in the septum of the ventricles between the sinus of the right and the base of the left ventricle, above which the aorta arose. The trunk of the pulmonary was much larger than its orifice, but its coats were very thin. The pulmonary veins entered the left auricle as usual. The ascending portion of the aorta was

unduly wide. The vessels given off at the arch were natural, and the ductus arteriosus was entirely closed.

The following were the dimensions of the heart:—

Girth of right ventricle externally—42 Paris lines = 94·5 mm. 3·72 En. in.
„ left „ „ 30 „ 67·5 „ 2·66 „

The right auriculo-ventricular aperture admitted a ball measuring in circumference 24 Paris lines = 54 mm. = 2·13 English inches. The pulmonary artery at its orifice had a circumference of 13 lines = 29·25 mm. = 1·15 English inches, but the connection between the conus arteriosus and the sinus of the right ventricle was only 7 lines in circumference = 15·75 mm. = ·62 English inches.

The left auriculo-ventricular aperture was 24 Paris lines in circumference = 54 mm. = 2·13 English inches. Aortic aperture connected with both ventricles, and had a circumference of 21 Paris lines = 47·25 mm. = 1·86 English inches.

The anterior wall of right ventricle when recent measured 6 Paris lines in thickness = 13·50 mm. = ·53 English inches; after maceration the same part measured 4 Paris lines = 9 mm. = ·35 English inches.

The posterior wall then measured 3·5 Paris lines = 7·89 mm. = ·31 English inches, and the anterior wall of the left ventricle was also 3·5 Paris lines in thickness.

1. In reference to this case, though no doubt could exist as to there being a cardiac defect of congenital origin, the precise nature of the anomaly was not diagnosed during life, and it seems remarkable that at least an approximately correct conclusion should not have been arrived at. The failure of diagnosis was, however, entirely due to the absence of any distinct murmur, till shortly before the death of the child. The small size of the aperture of communication between the sinus of the right ventricle and the conus arteriosus, and the circle of vegetations which surrounded that opening, makes it extremely difficult to explain such absence. It is true that the aorta communicated freely with the right ventricle, and in this way the blood may have so readily escaped from that cavity as to prevent the pressure on the opening into the conus arteriosus being sufficient to produce a murmur.

Dr. Barlow, of the Children's Hospital, tells me that when first the child was in the Hospital in Great Ormond Street, a slight murmur was heard at the base, but that it entirely disappeared after a time.

2. Dr. Farre,[1] in 1814, expressed the opinion that the cases in which there is constriction of the orifice of the pulmonary artery and the septum of the ventricles is deficient, so that the aorta communicates with the right ventricle, is the most common form of malformation; and the numerous cases which have been placed on record since that time entirely confirm the accuracy of his opinion. It is, however, now known that the obstruction at the outlet of the right ventricle is not, as he supposed, confined to the pulmonic valves. It may occupy several different situations, being either at the free extremities of the valves, at the fibrous zone to which they are attached, or at the commencement of the conus arteriosus or the point of separation between the conus and the sinus of the ventricle. Of these situations the first is probably the most common, while obstruction at the commencement or end of the conus arteriosus, as occurred in this case, is comparatively rare. It is also not usual in cases of serious obstruction at the outlet of the right ventricle to find the foramen ovale and ductus arteriosus imperforate. In this case these passages were however closed, and the peculiarity can only be explained on the supposition that at the time of birth the outlet for the blood from the right ventricle into the aorta was so free as to prevent any undue distension of the right auricle, and that a sufficient quantity of blood passed through the pulmonary arteries to allow the ductus arteriosus to be obliterated. Dr. Hunter, in describing a somewhat analogous case, suggested that the imperfection in the septum of the ventricles might be the result of the great amount of obstruction at the orifice of the pulmonary artery causing the blood to flow directly into the left ventricle, and so rendering permanent the opening in the interventricular septum. Meckel, on the contrary, supposed that the primary deviation from the natural process of development consisted in the imperfect closure of the interventricular aperture, and that the defect at the pulmonary orifice was due to the circulation being almost entirely diverted from the pulmonary artery. Such a theory is applicable to the rare cases in which the pulmonary orifice and artery are simply small, but affords no explanation of the more numerous ones in which the pulmonic valves and the parts adjacent are more or less seriously diseased. The former theory, therefore, is most probably the correct one; and it seems almost necessarily to follow that, provided the connection between the aorta and the right ventricle remains, the

[1] 'On malformations of the human heart,' p. 27.

aorta would gradually be drawn more over towards the right side, and the peculiar widening of its ascending portion, which so generally exists in cases of the kind, would be produced.

3. The peculiar chilliness which forms so striking a feature in cases of cyanosis dependent on malformation of the heart, has attracted the attention of most writers on the subject, and it seems to have been accepted as a well ascertained fact that the temperature of cyanotic children is below the normal standard. I do not, however, know that there are any conclusive observations on record to prove that this view is correct. In 1854 Dr. Farre made several observations upon a cyanotic child æt. 4, and though the temperature of the thermometer in the hand of the child rose only to 85°, whilst in his own it attained 98°, he found the temperature under the tongue both of the child and of himself to be 98°; and subsequently observations made on different days in August and September, when the external temperature ranged from 52° to 61°, and the temperature of the room from 59 to 70°, the temperature under the tongue was 99°, and of the hand from 74 to 98°, the pulse ranging from 108 to 96°, and the respirations from 28 to 32. In another case, in a man, æt. 22, the temperature of the room being 76°, the temperature of the hand was 98°, and under the tongue 100° and a fraction, or 2° above his own temperature. These facts appear to show that the temperature of the body attains in cases of this kind the normal standard, but that the process of cooling in the exposed parts is more rapid. I have myself in several instances made observations on the temperature of children and with very similar results. In the case of this child the temperature in the axilla was generally quite up to the normal standard, and it was somewhat higher than it should be on several occasions before the occurrence of spitting of blood. During the febrile excitement which attended the attack of measles the temperature was very high, reaching on the fifth day of active illness 104·9°, a temperature which slightly exceeded that of any other of the children who had measles in the hospital.

Dr. Barlow has favoured me with some observations which he made while the child was in the Great Ormond-street Hospital, and from these it appears that the temperature did not usually materially differ from the normal standard, though on two or three occasions it was unduly low. *May 2nd*, 1876.

22. *Dissecting varix of the left femoral vein.*

By C. Hilton Fagge, M.D.

The preparation was taken from the body of a woman, æt 67, who died, in Guy's Hospital, of bronchitis and emphysema, attended with considerable anasarca.

On cutting down upon the left femoral vein I found it in a remarkable condition. It was represented by a double channel, which extended for several inches, the intervening septum being thin, and perforated by a number of round apertures of communication. Each of the two tubes had a smooth lining membrane, and neither of them contained any thrombus. In the posterior attachment of the septum there was a third tube, much smaller than the others, down which a probe could be passed for a considerable distance. This had occasional imperfections in its walls, which gave the appearance of a row of horizontal bands passing across each of the larger tubes on one side. The several channels all opened below into the trunk of the vein, which was normal. How they terminated above could not be ascertained, the parts having been previously injured. The affected part of the vein was much thickened, irregular in outline, and adherent to the adjacent parts.

The right femoral vein was healthy.

Remarks.—It appeared to me evident that this affection was the result of a past attack of thrombosis of the vein, which had probably occurred after one of eight confinements that the woman had had. I am not aware, however, that such a condition has before been observed. I could not make out that one of the two tubes rather than the other represented the original channel of the vein. I am disposed to believe that they both were new formations.

In consequence of the analogy between the appearances presented in this case, and those which are observed in some of the more extreme forms of dissecting aneurysm of the aorta, I have called the specimen a "dissecting varix," but the name is not a very good one.

May 2nd, 1876.

23. *A case of disease of the aorta with malformation of pulmonary artery.*

By J. Burney Yeo, M.D.

This specimen was unfortunately greatly mutilated in removal from the body, as the *post-mortem* examination had to be made hurriedly and under peculiar circumstances.

It consists of the heart and a portion of the aorta taken from the body of a boy, æt. 10, who died under my care, in King's College Hospital, on the 4th of April.

It would seem to be a case of congenital malformation conjoined with recent inflammatory disease.

The aorta, about half an inch above the semi-lunar valves, appears to be greatly thickened and constricted, and its canal at the constricted part is nearly closed by vegetations. Just above the constricted part and to the right of it, there is a very remarkable pouch, the cavity of which is about the size of a hazel nut, and its walls which are firm and of uniform structure are about one quarter of an inch in thickness. It has a distinct, smooth, lining membrane. It was found filled with blood-clot. The heart was hastily separated from the large vessels before this condition of things had been discovered and this pouch was thus cut across.

On further investigation it was found that the inflammatory vegetations extended from the aorta into all the large vessels coming off from it, and that beyond the constricted portion seen in the specimen the aorta returned to its natural dimensions.

The aortic valves are perfectly healthy. The left ventricle is greatly hypertrophied. The pericardium contained several ounces of fluid and the visceral layer was covered here and there with rough patches of lymph.

There is also an abnormal condition of the pulmonary artery. It appears to come off as two trunks, from the right ventricle—the division taking place about three-quarters of an inch above the sigmoid valves. The posterior of these crosses the aorta transversely, and probably tended to press upon it, and contributed to the constriction.

The history of the case is as follows:—The boy was brought to the Brompton Hospital as an out-patient on the 5th of February, complaining of slight cough and great dyspnœa. The severe dyspnœa had been coming on for about three weeks—but he began to be ill a

week before Christmas, when he complained of chilliness and of pain in the left side, since then he has had a dry cough. *He had never complained of dyspnœa before this illness.* He had never had rheumatic fever or chorea, but he had had small-pox, and a very severe attack of measles when he was two years old. His father had had acute rheumatism twice.

When I saw him again on the 19th of February, the dyspnœa had considerably increased and I had him admitted into the children's ward in King's College Hospital. The physical signs, at that time, were these :—The area of cardiac dulness was increased every way. The impulse was strong and diffused, the apex beating in the 6th interspace about three quarters of an inch outside the nipple line.

A very loud, harsh, prolonged systolic murmur was heard all over the chest—most intense at the base of the sternum. It was propagated along the large vessels.

The respiratory sounds, anteriorly, were entirely drowned by the loud, cardiac murmur—behind a few sub-crepitant râles were heard at both bases and an occasional expiratory rhonchus. There was great pallor of the surface and great emaciation. The pulse was small and frequent, ranging between 120 and 160. With rest in bed in the hospital, the breathing became much more tranquil, and he seemed for a time to be mending, but there was always a remarkable daily oscillation in the temperature, ranging, with tolerable regularity, from 98° in the morning to 103° in the evening.

On March 31st, after he had been in the hospital about six weeks, scarlet-fever unfortunately broke out in the ward in which he was, and he had a mild attack. This was attended with increased distress of breathing so that he could no longer lie down in bed.

On April 3rd, the symptoms of the fever had disappeared, and his mother insisted on removing him from the hospital. The day after he died.

It is much to be regretted that this specimen suffered such mutilation in its removal, otherwise I believe it would have been valuable as an instance, almost unique, of combined congenital and acquired disease of the aorta, ending in an almost entire closure of the vessel. It is impossible that the abnormal conditions observed in the aorta could have owed their origin entirely to comparatively recent inflammatory action. It is equally difficult to believe that the state of things we see there is altogether congenital ; for on careful inquiry I find that prior to the attack of illness which came on just before

Christmas, this little patient went to school and ran about like other boys without suffering from any noticeable dyspnœa.

The most probable account of the case I take to be this:—There was originally a certain amount of congenital constriction of the aorta associated probably with the existence of the abnormal pulmonary artery and of the curious pouch we see on the aorta, and that when the acute attack of pericarditis came on, coincident with it the constricted part of the aorta became inflamed—the inflammatory action being determined at this part by the great amount of friction of the blood current against it—just as one sees a patch of endocarditis set up by the friction of a regurgitant current of blood against the wall of the ventricle. The inflammatory action excited at the constricted part extended thence into the adjacent vascular trunks, and these were lined with vegetations. It is difficult to understand how the circulation would have been maintained through so small an aperture as that which the aorta afforded; for its canal now appears to be almost completely blocked up. No doubt this condition must have arisen gradually—the calibre of the tube becoming slowly smaller and smaller. The contractions of the hypertrophied ventricle following one another in rapid succession continued for a time to supply the system with blood enough to maintain life; while the great emaciation and marked pallor indicated defective nutrition and a comparatively empty state of the arteries.

May 10*th*, 1876.

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24. *Congenital heart disease; two cases.*

By Thomas Barlow, M.D.

Albert D—, æt. 3 months, was brought to the out-patient department at the hospital for Sick Children. He was ricketty and marasmic. His diminutive size and slate-coloured lips were the only signs which raised the suspicion of congenital heart affection. He was under observation for seven months, during which time his nutrition greatly improved and with it a progressively increasing hypertrophy of the heart was observed. At no time was there any

murmur. There was no clubbing of finger ends. Death took place from an attack of acute bronchitis. The heart was found hypertrophied and dilated, the right being the larger of the two ventricles.

The foramen ovale was patent but valved, and probably had not transmitted blood after birth. The tricuspid orifice was normal. The pulmonary artery arose from the normal position, but was bigger and thicker than usual. After dividing, it was continued by what might be called the ductus arteriosus into the descending aorta.

This system had no connection whatever with the ascending aortic arch. It would appear that the descending aorta was formed by the persistence of the left fifth primitive arch.

To the right of and posterior to the origin of the pulmonary artery, *but also arising from the right ventricle* was the ascending aorta. It was smaller than the pulmonary artery. After giving off the two coronary arteries it was divided into the innominate, left carotid and left subclavian.

The left auricle and mitral orifice were natural.

The left ventricle did not give off any vessel but opened by a large aperture at the "undefended spot" into the right ventricle.

Some cases of the descending aorta arising from the pulmonary artery, that is, from the fifth left arch are recorded by Dr. Peacock, but the origin of the ascending aorta to the right of and posterior to the pulmonary artery, has not I believe been noticed before.

Case 2.—Frances B—, æt. 10 weeks, was brought to my out-patients at Great Ormond-street, July 9th, 1875. She was seen first by Dr. W. Allen Sturge who, in the course of examination, detected the heart disease, although there had been no sign directing attention thereto. In a week afterwards when I saw the child, there was, however, distinct though slight lividity of the nose, lips, and tongue.

There was no thrill to be felt over the cardiac region but there was a heaving impulse below the left nipple, and there was a loud systolic murmur heard down the sternum and at the apex and into the axilla. It was as loud at the apex as at the ensiform cartilage and decidedly louder than at the base. If the murmur had been heard in an adult, one would have pronounced it that of mitral regurgitation. The child had stomatitis and aphthæ about the genitals, looking very puny, and on one side had, I believe, a displaced lens with partial wasting of the globe, and internal strabismus.

It died, probably from asthenia, before it was twelve weeks old.

The foramen ovale was widely patent. The tricuspid orifice was absolutely closed, but its position was marked by a white line probably a cicatrix. The venous blood must have passed therefore entirely through the foramen ovale into the left auricle, which was dilated.

There was a large mitral orifice and vegetations on the curtain of the valve.

The left ventricle was hypertrophied and dilated. From it some of the blood was driven into the aorta, and some through a small aperture at the "undefended spot" into the right ventricle.

The right ventricle was exceedingly small. No trace could be detected of the tricuspid valve beyond the cicatricial (?) line before referred to in the right auricle.

From the right ventricle in the normal position was given off the the pulmonary artery.

There was a small frænum joining the united extremities of two of the pulmonary valves to the wall of the artery.

The heart was removed under difficulties, and the vessels were cut too short for complete identification. The aorta after giving off the two coronary arteries was continued up into a sac which terminated in an impervious cord and a minute vertical vessel. From the side of this aortic sac was given off a small arch turning to the left. This arch had two ascending branches and was then probably continued into the pulmonary artery. At all events the pulmonary artery gave off *before bifurcating*, a branch going towards the ascending aortic arch and a *large descending branch* presumably taking the part of the descending aorta.

There was some collapse of the lower lobes of the lungs, but the other viscera were natural.

The specialty of this case was the closure, probably by fœtal endocarditis, of the tricuspid orifice.

May 16th, 1876.

IV. DISEASES, ETC., OF THE ORGANS OF DIGESTION.

(A) TONGUE AND DIGESTIVE CANAL.

1. *Ulceration of the stomach from sulphuric acid taken by mistake.*

By Thos. B. Peacock, M.D.

The subject of the case was a man, æt. 42, of very intemperate habits. He swallowed about a table-spoonful of sulphuric acid by mistake about five weeks before his admission into the hospital. Vomiting was promoted at the time, but no antidote was used, and he had suffered severely ever since. Two weeks after the occurrence he brought up a large quantity of blood, and while in the hospital he had incessant vomiting. There was a circumscribed tumour in the epigastrium, which was sometimes large, tense, and dull on percussion; at others smaller, softer, and resonant. The tumour was generally largest and most tense before the attacks of vomiting, and partly subsided after the contents of the stomach were freely evacuated. He survived for about three weeks after his admission.

The mucous membrane of the lower part of the œsophagus, and in various places in the greater curvature of the stomach, was deeply and extensively ulcerated, and there was some contraction of the pyloric orifice.

The kidneys were somewhat granular.

October 19th, 1875.

2. *Epithelioma of tongue involving the lower jaw. Removed.*

By Christopher Heath.

E. J—, æt. 52, was admitted into University College Hospital under Mr. Heath's care in September, 1875, with very extensive cancerous disease of the tongue and sublingual structures. In January, 1874, was noticed a swelling of one of the submaxillary glands, and soon after a sore beneath the tongue. The sore healed, and the gland subsided under treatment. In September, 1874, the gland began again to swell and at last broke. At the same time he found that he had difficulty in articulating, as the tongue was fixed to the floor of the mouth, and eventually the tip of the tongue became fixed to the jaw. This was temporarily relieved last April by an operation undertaken in a London Hospital. On admission the patient was unable to protrude his tongue or move it in his mouth, the saliva constantly trickled away and articulation was very imperfect. He complained of great pain in the occipital region, but of none in the tongue. The gums of the incisor region were swollen and ulcerated, and the teeth loose. The tongue was fixed to the back of the jaw, which was softened. All the tissues beneath the tongue were indurated, but the skin was not involved.

On Sept. 29th Mr. Heath removed the tongue, centre of the jaw, and all the sublingual tissues, by dividing the skin in the middle line, then sawing the jaw through on each side, and, having isolated the tongue somewhat on each side, by enclosing the whole of the disease with the wire of the galvanic écraseur. The patient made a good recovery, and was shown to the Society.

The parts removed consisted of the middle three inches of the lower jaw, nearly the whole of the tongue, and the sublingual muscles and glands *en masse*. At the posterior end, the mass measures two and a half inches in depth, and slightly more from side to side. The tongue appeared to be healthy, except at the anterior part; and on the left side, just behind the tip, was a nodule of the size of a pea. Beneath the tongue was a mass of a yellowish white firm tissue, with a granular surface, which was continued quite up to the cut margin. This tissue, on microscopical examination, proved to be epithelioma.

Examined microscopically.—The gums and the tissues at the root of the tongue, even to the edge of the part, are found to be epitheliomatous. Characteristic "globes" are only very small and few in number; but collections of large epithelial cells, of varied shapes, with clear distinct nuclei, are very abundant and characteristic. The growth is infiltrating the muscles at the root of the tongue; but the tongue itself, except at the tip, is free. *November 2nd*, 1875.

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3. *Cancer of rectum for which colotomy was performed two years and nine months before death.*

By Christopher Heath.

Charlotte T— was admitted into the hospital under Mr. Heath's care in January, 1873, with a history of difficult defæcation for nearly a year, flattened fæces, and occasional passage of blood. The rectum was completely blocked up with hard cancerous growth, of which a portion of the size of a walnut protruded from the anus, through which the little finger passed with difficulty. Mr. Heath performed colotomy in the left loin on January 18th, 1873, and the patient made a good recovery, being discharged on March 18th. The patient continued in fair health, but the disease steadily increased, perforating the vagina, and protruding both from the anus and vulva, and she entered one of the cancer wards of the Middlesex Hospital, under the care of Mr. Lawson. There she remained until her death, which took place on October 17th, 1875.

At the *post-mortem* examination the cæcum, ascending and transverse colon were found to be notably small; the last-named portion of the intestine dwindling in size to its termination at the splenic flexure, where its calibre was reduced to fully one third of the average, even when it was fully distended. At the splenic flexure the bowel was firmly adherent to the abdominal parietes, and at this point it opened externally by an artificial anus in the loin. Thence downwards to below the brim of the true pelvis the large intestine was reduced to a cylindrical structure no larger than the fore-

finger; the canal, however, was still pervious. The lower part of the rectum was enlarged, and the seat of new growth, which almost completely blocked the canal, and appeared at the canal outlet as a large fungating cauliflower-like mass. On laying open the vagina the growth was seen to have invaded the wall of this structure, and to have ulcerated through its posterior wall, so that a large cloacal chamber had been formed by the rectum and vagina.

The uterus was free from invasion, although the vaginal wall had ulcerated completely up to the os uteri. Numerous fibroid tumours occurred in the uterine wall. The liver was enlarged, the greater part of its left lobe, and a portion of the right, being the seat of a firm, dense, white, lobulated mass, projecting on the surface of the organ, where the capsule was thickened and puckered. The remaining viscera were small but healthy.

Microscopical examination showed that the rectal growth was a *cylinder-celled epithelioma*. The mass in the liver presented also tubes and cylinders of columnar epithelium, surrounded by a large quantity of imperfect fibrous tissue, infiltrated with small fatty granules.

November 2nd, 1875.

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4. *Two specimens of distension-diverticula of the small intestine.*

By C. Hilton Fagge, M.D.

The first specimen was taken from the body of a woman, æt. 57, who died in Guy's Hospital on July 21st, 1873. She had been seized with symptoms of acute obstruction of the bowels thirteen days before, after taking a dose of compound jalap powder. She was under the care of Dr. Bright, of Forest Hill, who sent her up to the hospital.

On admission she was cold and collapsed, and it was evident that she would die unless relief could be given her. I therefore asked Mr. Davies-Colley to see her; and he performed an exploratory

operation. The cause was then discovered to be the inclusion of a very small knuckle of intestine in the right femoral ring. It was withdrawn, and was found to be of a deep chocolate colour. The small intestine above also was chocolate-coloured, and it was extremely distended. She did not rally, and died the same evening.

Several coils of intestine had to be drawn cautiously out of the abdominal cavity before the seat of the obstruction could be found, and two rounded diverticula were then noticed, projecting from the mesenteric attachment of the gut, and indeed passing between its two peritoneal layers.

The *post-mortem* examination was made by Dr. Goodhart. The two diverticula were found to be situated a few inches down the jejunum, at a short distance from one another. One was from 1 to $1\frac{1}{2}$ inch in diameter, the other a third of that size. They were irregularly puckered on the surface, and were evidently protrusions of the intestinal mucous membrane into the space between the two peritoneal layers of the mesentery. The part of the bowel which had been strangulated was about half way down the small intestine.

Just above the cæcum, in the right lumbar region, a coil of small intestine was contracted and matted together by old adhesions, the course of the bowel being very tortuous, but there was no decided constriction nor any sharp twist.

The second specimen was taken on October 26th, 1875, from the body of a man, who was brought into the hospital dead. The cause of death was not very apparent, but the muscular tissue of the heart was soft and brown. The intestines formed at two parts of their course agglomerations exactly like those which are so often seen in cases of scrotal herniæ, portions of the bowel which had for a time been enclosed in the sac becoming fused together. No hernia, however, could be discovered. The larger agglomeration was as big as a cricket-ball, and it contained several diverticula which protruded into the mesentery, between the layers.

Remarks.—The diverticula in these cases were entirely different from those relics of a fœtal structure which are sometimes found projecting from the free edge of the ileum. I have called them distension-diverticula, because in the first case everything seemed to indicate that they had been formed, during the patient's fatal illness, by the pressure of the intestinal contents. It did indeed strike me as remarkable that the pouches remained after the intestines had been emptied, and did not shrink and disappear, as other effects of

rapid distension in the organs of the human body commonly do. The second case, in which similar diverticula were found in a part of the intestine agglomerated by old adhesions, does not throw much fresh light upon their cause, for the patient may very probably have suffered from an attack of ileus and intestinal distension at some former period. *November 16th*, 1875.

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5. *Tubercular lupus of tongue, palate, and gums.*

By W. Fairlie Clarke.

Mr. Fairlie Clarke exhibited a specimen of tubercular lupus of the tongue, palate, and gums, which was taken from a young man who died in the West London Hospital under the care of Mr. Teevan.

The patient was a bricklayer, æt. 18, and he was under observation at the hospital from December, 1874, until April, 1875, when he died. There was no family history of phthisis, syphilis, or cancer. The skin of his face was perfectly free from any blemish. He had no cough, and none of the physical signs of phthisis. On examining the interior of his mouth after death the whole of the mucous tissues were found to be congested, enormously thickened, and velvety on the surface. The soft palate had completely perished, and there was a deep ulcer on the left side of the tongue, with some slight ulceration on the corresponding portion of the gum. The thickening of the mucous membrane covering the left side of the lower jaw was so great as almost to simulate a growth connected with that bone, and the mucous lining of the corresponding antrum was also very much thickened. The epiglottis was somewhat thickened, but not ulcerated. The parotid and submaxillary glands on the left side were much enlarged, but the lymph-glands were not affected. The mucosa of the nose was thickened and ulcerated.

As the case was evidently an uncommon one, it was submitted to Sir J. Paget six weeks before death, and he diagnosed it as tubercular lupus.

Portions of the tumid mucous membrane were examined microscopically, both by Dr. Ed. Sparkes and by Mr. Butlin, and it was found that the interstices of all the affected tissues were infiltrated with small round cells (leucocytes).

No tubercles were found in the lungs at the *post-mortem* examination. The patient died of exhaustion, induced by the backward pressure of the local disease, and by his inability to swallow food. For some days before his death he was unable to open his mouth in the least degree, and was kept alive by enemata given *per rectum*.

Primary tubercular ulceration is very seldom seen in a mucous membrane, and, among the ulcers which affect the tongue, those which are of tubercular origin are extremely rare.

November 16*th*, 1875.

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6. *Two cases of congenital malformation of the pharynx and œsophagus.*

By Howard Marsh for Dr. Ilott.

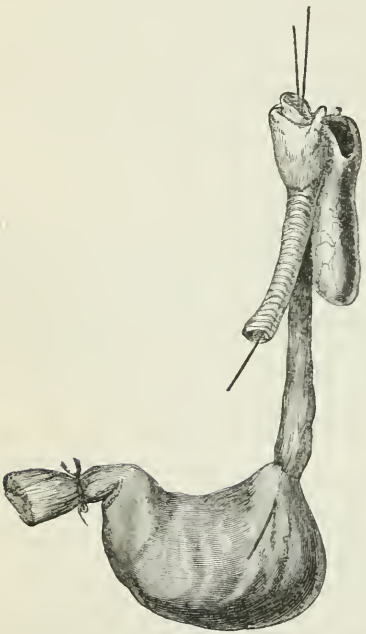
These two remarkable instances of congenital malformation occurred in the infants of patients who came under the notice of Dr. W. T. Beeby and myself, within three weeks of one another, in October and November, 1875.

Case 1.—A. S—, æt. 24, wife of a gentleman's gardener at Bickley, near Bromley, was delivered on October 13th, 1875, of a female child. The birth was premature, occurring at about the eighth month of pregnancy. Respiration was established with difficulty. The child suckled very imperfectly, though the mother had a full breast of milk. On attempting to feed it with milk and water from a spoon swallowing took place, but after an interval varying from a few seconds to two or three minutes regurgitation ensued through the mouth and nose, and the child became livid about the lips. Attempts to nourish it *per rectum* also failed. After the first passage of meconium no action of the bowels took place. The child became more feeble and emaciated, and died on October 17th, four days after birth. An opinion was given that some congenital obstruction

was the cause of death, and permission for a *post-mortem* examination was obtained. This was made on October 18th.

The lungs were imperfectly expanded and the tubes filled with muco-purulent fluid. The heart was of normal size, foramen ovale open; the stomach and small intestines were quite empty, but distended with air; the large intestine contained a considerable quantity of meconium; the pharynx, trachea, œsophagus, &c., were removed together. On examination the pharynx was found to end in a *cul-de-sac*, a little below the level of the cricoid cartilage. The œsophagus terminated on the posterior surface of the trachea, about half way between the cricoid cartilage and the bifurcation. On making an opening through its coats, just above its cardiac orifice, a probe could be passed upwards into the trachea (*vide* Woodcut 5).

Woodcut 5.



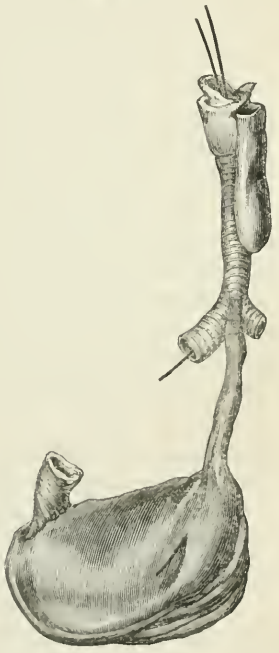
Case 2.—E. T—, æt. 40, the wife of a labourer, at Pope Road, Bromley Common, was delivered of her fifth child on November 4th, 1875. The infant was at full term and well developed. On the day following, when the child was put to the breast, it was noticed that after a short interval, about thirty seconds, the milk regurgitated through the mouth and nose, and the infant became livid about the lips. Meconium was passed in small quantities on this and the day following. From this date until its death, on November 10th, nothing passed through it. I saw the infant on November 6th, and had it fed in my presence. After a short interval the milk mixed with frothy mucus regurgitated through the mouth and nose. On auscultation air was heard entering both lungs, but feebly towards the bases, mingled, especially on the right side, with crepitation. I gave an opinion, based on my knowledge of the malformation in the previous

case, that there was a congenital obstruction, which would probably prove fatal. The infant progressively emaciated, and its breathing became more impeded until the 10th of November, when it died, having lived six days.

Post-mortem examination on November 12th.—Body emaciated; very little subcutaneous fat. The inferior lobes of both lungs were completely solidified from pneumonia, non-crepitant, solid, friable; the bronchi filled with a purulent fluid, which oozed from the cut surface on pressure. The upper lobes were crepitant, spongy, and of a bright red colour.

Heart healthy. Foramen ovale quite open. Liver very dark coloured, full of blood; umbilical vein patent, containing a little fluid blood. Meconium in upper part of small intestine and in large, also in vermiform appendix.

Woodcut 6.



Tongue, larynx, pharynx, trachea, œsophagus, and stomach removed. On examination the pharynx was found to end in a *cul-de-sac* a quarter of an inch below lower border of the cricoid cartilage (*vide* Woodcut 6). The œsophagus could be traced up to the bifurcation of the trachea, where it seemed to terminate; a few muscular fibres continuous with those of the œsophagus being traced along the posterior surface of the trachea to the pharyngeal pouch. The distance from the termination of the pharynx to the commencement of the œsophagus is a little more than half an inch, so I think it may be safely assumed that no communication could have been effected by art between the two. The specimen is also interesting from an abnormal disposition of the great arterial trunks, the right subclavian artery arising last from the descending portion of the arch of the aorta on the left side, and running behind the trachea and rudimentary part of the œsophagus to reach the right side: the two carotids being also given

off by a common trunk, which ascends a little way on the trachea, and then bifurcates into the right and left carotid.

Remarks.—These two cases of congenital malformation are of great interest, not only from a clinical point of view, but more especially as regards the development of the parts in the fœtus.

In 'Quain's Anatomy' it is stated that the œsophagus and pharynx are formed from the upper part of the primitive alimentary canal; that the mouth is developed by a depression of the outer surface of the embryo above the first branchial arch, and, together with the tongue, is at first separated from the throat by a partition, which soon gives way. In like manner the anal orifice does not at first exist, but is formed by invagination of the outer surface, and opening of communication between it and the intestine. The trachea and lungs appear as protrusions in front of the œsophageal portion of the alimentary canal. These primitive tubercles are visible in the chick on the third day of incubation, and in embryos of mammalia and man at a corresponding period. Their internal cavities communicate with the œsophagus, and are lined by a prolongation of its inner layer. At a later period they are connected with the œsophagus by means of a long pedicle, which forms the trachea, while the bronchi and air-cells are developed by progressive ramifications of their internal cavity in the form of cæcal tubes, after the manner of the ducts of glands.

It seems, then, that at a very early period the œsophagus and trachea are continuous with one another in some portion of their length, and that owing to some arrest of development a permanent communication between their cavities is left. Why this communication should take place at different points in the cases reported I cannot say. If, too, the pharynx and œsophagus are formed both from the upper part of the primitive intestine, it is difficult to understand the gap existing between the two.

In the 'Cyclopædia of Anatomy and Physiology,' in the article Œsophagus, it is said—"It sometimes happens that the œsophagus is congenitally deficient, terminating above in a *cul-de-sac*, the inferior extremity of the pharynx also terminating in the same manner; this is usually associated with an imperfect development of the oral cavity and of the lower jaw, the latter being in part or wholly deficient." Sir A. Cooper has recorded a case in which the œsophagus was altogether wanting, and the stomach was without a cardiac orifice. The child lived eight days. On referring to the past numbers of the Pathological Society's 'Transactions,' I find three cases

recorded, resembling or slightly differing from my own. In vol. iii, for the years 1850-51, Dr. P. B. Ayres records the case of an infant that lived eleven days. From the time at which it was first put to the breast it was observed that on every occasion after it was fed regurgitation of milk took place by the mouth, accompanied by convulsive movements and blueness of lips, and other symptoms of impending suffocation. A bougie passed down the œsophagus was arrested at some distance from the mouth. Meconium was passed in small quantities. Nutritive enemata were employed, but the child became more and more emaciated till its death.

The autopsy showed that the œsophagus commenced below the pharynx which was of natural size, but soon bulged into an elongated sac, which about an inch lower terminated in a *cul-de-sac,* a few fibres being continued downwards, forming a flattened imperforate band in close union with the posterior wall of the trachea. Just above the origin of the bronchi the œsophagus again enlarged,and terminated naturally in the stomach. A probe was passed from the stomach upwards and emerged at the larynx. The communication between the trachea and œsophagus was found to be by a slit-like opening on the posterior wall half an inch above the bifurcation.

In this case the distribution of the great vessels was normal, but the ductus arteriosus was nearly as large as the trunk of the aorta, and remained pervious, although the child had lived eleven days. The foramen ovale was not examined. Dr. Ayres mentions that he had referred to the works of Geoffroy St. Hilaire and Otto on congenital malformations, but was unable to discover any reference to a similar condition of parts.

This case nearly resembles my first, but differs in that there seemed to be a division of the pharynx and upper part of the œsophagus, and that the lower end of the *cul-de-sac* extended further downwards, and was formed by the upper part of the œsophagus and not by the termination of pharynx, as will be seen on reference to the plate accompanying his paper.

In vol. vii, p. 52, will be found a case by Dr. J. W. Ogle in which the trachea and œsophagus are the seat of congenital malformation. The infant in this case was healthy when born, and sucked vigorously; but most if not all the milk returned through the nares. It became convulsed before death, which occurred on the fourth day from birth.

Here the œsophagus was obliterated one inch from its pharyngeal opening, the upper part being very dilated. Below the obliterated

part there was an opening at the anterior surface of the œsophagus, which communicated directly with the larynx, at a point a quarter of an inch above the bifurcation of the latter into the bronchi, and hence the stomach and œsophagus were only continuous with the mouth by means of the glottis. This opening was valvular in form.

The next case will be found in vol. viii, p. 173, of the Society's 'Transactions,' reported by Dr. Ogier Ward. The subject was a male infant, born at full term; previous child had cleft palate. Immediately after birth a quantity of thick gelatinous mucus flowed from the nostrils, producing attacks of dyspnœa. At every attempt to feed or nurse the infant, milk regurgitated. A bougie passed was arrested five inches from lips. Child died on twelfth day. At the autopsy the pharynx was found to form a *cul-de-sac*, from which proceeded a thin band of muscular fibres to the bifurcation of the bronchi, from which point to the stomach the œsophagus regained its natural size and appearance. On passing a probe through the stomach it entered the trachea by a valvular opening just above the bronchi. Heart was of normal size; foramen ovale open. This case exactly resembles the second one that came under my notice.

The only other instances that I can find are one by Povis ('Archiv Univ. de Méd.,' ccxvii, 421, in Sydenham Society's 'Biennial Retrospect,' 1871-72, p. 153. The child lived two days. The symptoms exactly resembled those in the other cases. Upper part of œsophagus terminated in a *cul-de-sac* $2\frac{1}{2}$ centimètres below glottis. The communication between the œsophagus and trachea took place at a point corresponding to the bifurcation of the bronchi. In this case traces of milk were found in the stomach, which must have found its way through the trachea and opening, thence into the œsophagus. The other is related by Mr. Annandale in the 'Edinburgh Medical Journal' for January, 1869, p. 598. The gullet terminated in a *cul-de-sac* above the bifurcation of the trachea, again became pervious at the stomach and for some distance above. Lower portion of œsophagus opened into the trachea below the *cul-de-sac*.

The cases above related are all the instances I have been able to meet with of this rare and interesting malformation. The infants died at a variable period from their birth of two to twelve days. The diagnosis is easy if the attention has once been directed to the existence of such a state of parts. The treatment, on the contrary, seems hopeless, as the direction of the upper and lower parts of the digestive canal is not directed in the same line owing to the disten-

sion of the upper part and the inclination of the lower towards the trachea; besides, the gap between the parts of the tube is often considerable—as much as half an inch. Owing to the rarity of deformities of the upper part of the alimentary canal, even as compared with those of the lower (the varieties of imperforate anus) it will not often fall to the lot of a practitioner to consider the feasibility of any such proceeding. *November 16th*, 1875.

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7. *Ulceration of the duodenum; extension into the portal vein; hæmorrhage.*

By S. O. Habershon, M.D.

Celina T—, æt. 30, a married woman, was admitted into Guy's Hospital in September 1875. She had enjoyed good health till twelve years previously, when she fell from a railway carriage. Fits afterwards came on, and a year before admission she had rheumatism. Her illness dated from a miscarriage, about July, and from that time she suffered from pain in her side. Three weeks before admission she had severe rigors, which, with increased severity of symptoms, lasted six hours, and a fortnight later she vomited about three pints of blood. The patient was emaciated, anæmic, and sallow; there was severe pain in the left side and at the apex of the left lung, and there was much abdominal pain and distension. The hæmorrhage from the stomach recurred on September 19th, and again on October 6th, when a large quantity was rejected—several pints. Pitchy motions also were passed. She shortly afterwards sank.

The lungs were healthy, but there was a small quantity of recent lymph effused at the base of the right lung. The heart was healthy. The mucous membrane of the stomach was mammillated. About an inch beyond the pylorus there was a large ulcer with a depressed circular margin, but on the side towards the fissure of the liver was a large sloughy excavation; the whole of the coats of the intestine were destroyed, and the cavity reached to the fissure of the liver. The ulceration and sloughing had entirely destroyed the common

bile duct and the hepatic duct; the remains of the latter were on the floor of the ulcer. The vena portæ was laid bare, and an irregular ulcerative opening had been made where the vein was situated above the pancreas, from which fatal hæmorrhage had taken place. The hepatic artery was obliterated, the remains being visible at the ulcer. The gall-bladder contained some bile. The inflammatory action had extended into the portal canals, the vein was dilated, filled with bile-coloured clot, and there was much fibroid thickening in the course of the vein and in Glisson's capsule. The adjoining part of the liver to the right side contained an irregular abscess, and there were several smaller ones in the organ. One small abscess reached the surface, but adhesion with the stomach had prevented extravasation. The abscess appeared to be in the course of the portal vein. There was some general peritonitis, and a collection of pus in the pelvis. The left ovary was replaced by several cysts containing red grumous fluid, and was sloughy, and the right was enveloped in the same mass. *December 7th*, 1875.

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8. *Perforating ulcer of the stomach.*

By J. C. Thorowgood, M.D., for Dr. Wilton.

H. T—, æt. 20, a gardener, had been under the occasional care of Dr. Wilton and Mr. Bosworth, at Sutton, on account of dyspepsia.

The attacks were characterised by severe pain and vomiting of food, but he had never been ill enough to lose one day's work, and had never vomited blood.

January 9th, 1876, he came to the surgery, in the evening, complaining of colicky pains in the stomach.

By morning these pains were better, and he did his work in the garden during the whole day.

At night he again came to the surgery in much pain, which increased till at 10.30 he was in a state of collapse, and at 2 o'clock he died.

Post-mortem examination.—Body well nourished; general peri-

tonitis; much fluid in the cavity of the abdomen. On pressing the stomach fluid was seen oozing from a circular ulcer near the pylorus.

The ulcer had round thickened edges, and was of a size sufficient readily to admit the tip of the finger. No other point of ulceration could be observed.

April 4th, 1876.

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9. *A case of chronic obstruction of the small intestine, due to old adhesions connected with caseous disease of the mesenteric glands.*

By C. Hilton Fagge, M.D.

Caroline M—, æt. 9, was admitted into the Clinical Ward of Guy's Hospital, under my care, on April 7th, 1876. She said that she had enjoyed good health up to the last fortnight and that she had had sufficient food; but it appeared that her father was in the workhouse, and that her mother had to go out charing for the support of herself and two daughters, in which object, however, a son assisted her. Careful inquiry also elicited the fact that she had at various periods complained of pain in the abdomen.

Thirteen days before her admission she was seized with severe pain in the abdomen and with retching. Her bowels were open on that day, but they were confined for the eight following days. On the 9th day (April 3rd) she was seen by a medical man, who ordered oleum ricini: after this a slight evacuation from the bowels occurred. She was constantly sick for the first week, but not so much so during the second week.

On admission she was found to be fairly grown for her age, but much emaciated. Skin cool. Pulse 112, small, and very feeble. Abdomen distended, universally tympanitic, tender. Coils of the intestine visible through the parietes, sometimes much more distinctly than at other times. While one was looking at the abdomen, and manipulating it, a kind of spasm would come on, which seemed to be little if at all painful, during which the intestine formed a series of rounded prominences, separated by deep grooves.

On the 7th she was ordered to take the tincture of opium in ɱv doses every four hours, to have an enema, and to have milk diet.

On the 8th it was noted that after the enema several large scybalæ came away. In the afternoon the vomited matters were found to be stercoraceous, frothy, and like a loose stool.

On the 9th the enema was repeated, but it did not bring away anything. She had stercoraceous vomiting again.

10th.—No more vomiting. Temperature 98°; pulse 120, feeble; respiration 14; urine acid, of sp. gr. 1010, containing neither albumen nor sugar.

11th.—Temperature 99·2°; pulse 116; respiration 14. She vomited again yesterday. The bowels were not open. The breath and body were noticed to have a very foul odour.

From the first it appeared to me that this case was one of chronic obstruction of the small intestine, and that it probably belonged to a class to which I had drawn attention in a paper in the 'Guy's Hospital Reports' for 1869, under the name of "Contractions." I then showed that whereas the jejunum and ileum were scarcely liable to true stricture, they not unfrequently became obstructed as the result of adhesions binding their coils together, or to the abdominal walls. I related several instances in which simple chronic peritonitis or malignant disease of the peritoneum had led to such a result; and three other cases in which enlargement of the mesenteric glands had played an important part in bringing about obstruction of the bowels. Considering the early age of my patient, I was disposed to think that previous disease of these glands —*tabes mesenterica*—had probably been the starting-point of the mischief.

One feature of the case, which weighed greatly with me as indicating the nature of the disease, was the marked way in which the distended coils of intestine were seen through the abdominal walls. In my paper in the 'Guy's Hospital Reports' I had suggested that it is only in the chronic form of obstruction that peristaltic movements of the gut are visible, the reason being that an hypertrophy of their walls is requisite for the production of this appearance. And since that time I have failed to meet with any exception to the rule I then laid down. I have never yet seen coils of intestine rolling over one another in cases of acute strangulation; nor do I think that their outlines can be so plainly seen through the parietes in any cases of this kind as in those which run a chronic

course. It has been suggested that the emaciation of the abdominal walls caused by long-standing disease may account for the distinction: but I do not think that this is an adequate explanation. I am not, however, altogether satisfied with my own way of accounting for it: I have latterly thought that the absence of visible movements in acute cases might perhaps depend upon the paralysed state of the intestine.

In the case now under consideration I particularly noticed that day after day the coils of intestine occupied precisely the same positions in the abdomen, although the contractions which made them visible were spasmodic and intermittent. This led me to think that the serous cavity was probably closed by adhesions; and that, if so, the case was a very favorable one for the performance of the operation of making an artificial anus in the groin. On the 12th, therefore, I asked one of my surgical colleagues to see the patient. He, however, was of opinion that it would be advisable that the injection of a large enema into the rectum should first be tried.

The girl had vomited once more on the previous night. Pulse 116; respiration 12; temperature 97·6°. At 5 p.m. an enema of 25 ounces of tepid water was administered very cautiously. As soon as she complained of severe pain the pipe was withdrawn; it was introduced only just within the anus. The fluid was retained for a few minutes, and then almost the whole of it was returned, and with it a few lumps of light coloured fæces. She did not at the time appear to be the worse for the operation; but soon afterwards she was found to be collapsed, and in spite of all efforts to restore warmth she died at 2 a.m. of the 13th.

The *post-mortem* examination was made the same afternoon by Dr. Goodhart.

The lungs and heart were healthy; the glands at the bifurcation of the trachea were rather large and were converted into a mass of putty-like substance.

The abdomen was not much distended. The surface of the peritoneum was injected. The intestinal coils were glued together by recent lymph, and there was a turbid puriform fluid to the amount of 5 or 6 ounces in the lower part of the abdominal cavity. This was found under the microscope to contain broken-up fibres and bodies which were probably starch granules; it was evidently chyme mixed with pus. The former seemed to have escaped through a minute aperture in a coil of the jejunum lying near the spine. The

tissues around this were blackened; and on manipulation a pultaceous matter came freely through it.

Between the sigmoid flexure and the ileum there were old and organized adhesions, with a caseous gland in the angle between them. Another old adhesion between the omentum and the small intestine existed in the left hypochondriac region. The liver also was firmly adherent to the diaphragm. When the distended part of the bowel was displaced, a great length of contracted bowel—all small bowel—was seen lying in the right lumbar region and across the spine. The cæcum also was contracted; it was in an unusual position, bound up beneath the anterior edge of the right lobe of the liver, with its appendix closely adherent to that organ, and a caseous gland in close proximity. The right iliac fossa was empty, and lined with a smooth layer of peritoneum. From the cæcum the large intestine ran its usual course: it was somewhat adherent to the parts near which it passed: it was closely contracted, containing only a thin coating of yellowish pultaceous matter.

Subsequent examination of the small intestine showed that the part of it which was contracted consisted of about half the ileum from the cæcum upwards. This was bound down to the spine by thickening and shortening of its mesentery, all the glands in which were much enlarged and caseous. At the spot where the bowel became suddenly distended a knuckle of it was fixed by old adhesions to the under surface of the liver, forming a somewhat sharp angular bend. This loop also seemed to have been dragged upon and stretched, and the other coils to have been slightly twisted round it.

The result of the autopsy thus accorded fairly well with the diagnosis that had been given during life, except that the peritoneal cavity was not found closed by universal adhesions.

The only remaining question is as to the cause of the sudden fatal collapse. Some years ago a patient under my care was considered to have died from the shock of a large injection of seven pints, too rapidly administered by one of the resident pupils. But in the present case the quantity thrown up was comparatively small, and it was thrown up very cautiously; and I have little doubt that the supervention of the fatal symptoms at so short an interval afterwards was a mere coincidence.

April 18th, 1876.

10. *A case of communication between the vermiform appendix and the rectum.*

By Jeremiah McCarthy.

This specimen was obtained from the body of a woman who died in the London Hospital, of broncho-pneumonic phthisis.

The vermiform appendix was firmly adherent to the back of the first part of the rectum, the mucous membrane of which, opposite to the adhesion, was perforated by two small holes, which opened into a cavity in the submucous tissue about the size of a small bean. Into this cavity the end of the vermiform appendix projected in a nipple-like manner, and a small probe could be passed through the appendix into the rectum. The mucous membrane of the cæcum and ileum was ulcerated as is usual in broncho-pneumonic phthisis, but that of the appendix was perfectly healthy. It is probable, therefore, that some substance, fæcal or otherwise, had at some time become impacted in the vermiform appendix, and there set up inflammation; that, fortunately for the patient, adhesion to the rectum resulted, and subsequently the coats of the rectum and end of the appendix having ulcerated, an abscess formed in the submucous tissue of the rectum, the contents of which, with the exciting cause, were finally discharged into the rectum through the perforated mucous membrane. *April* 18*th*, 1876.

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11. *Obturator hernia in a female, causing chronic intestinal obstruction; death from suppurative peritonitis.*

By James F. Goodhart, M.D.

Susan G—, æt. 65, a married woman, was admitted into Guy's Hospital on the 26th of May, 1875, under the care of Mr. Bryant. She had enjoyed very good health till 1871, when one morning after

considerable exertion she experienced great pain in the left side, and was able to sit down only with difficulty. The medical man said she had a hernia, but that it had gone up. Her health remained indifferent, but she was able to get about till six months before her admission, when she noticed that she had passed no motion for nine days. Purgative draughts acted upon the bowels very freely, but she was very sick also. She then continued well until her admission. At that time no motion had passed for ten days, and she had vomited continually. She looked very unhealthy, being much emaciated and yellowish. However, no growth could be detected in the rectum. She was cold and collapsed. The abdomen somewhat distended, with visible coils of small intestine and peristalsis. She complained of great abdominal pain. Opium was administered, and warm fomentations were applied to the abdomen, and she became much easier, and four days after the bowels acted twice spontaneously, and subsequently she had repeated loose evacuations.

She left the hospital well, except for extreme emaciation, on June 15th, 1875, nineteen days after her admission. She was readmitted on December 26th, of the same year. She had remained quite well after leaving the hospital till December 3rd. For the last three months she had been in much reduced circumstances.

On December 3rd she was again violently sick, and had much pain in the left side of the abdomen, increased on passing a motion. The bowels were confined and fæces small.

She had now a double femoral rupture easily reducible. Her abdomen was natural, and a rectal and vaginal examination showed nothing abnormal. She was again placed under the influence of opium, and the sickness ceased and the bowels acted.

On January the 6th, however, the symptoms returned. She was again sick, had much pain in the left side of abdomen, and the temperature rose to 101° F. From that time she gradually sank. The bowels were not again opened, the abdomen became tympanitic, and the vomiting only ceased, with increasing exhaustion, a few days before death, which took place on February 4th. She had at no time anything like obturator pain, and though all the regions of hernia were examined carefully, nothing was noticed to suggest the disease.

Autopsy.—The body was very emaciated; there was no marked distension of the abdomen; the peritoneum was injected all over. A little

pus was smeared over the coils in the neighbourhood of the cæcum, and in the pelvis two ounces of pus or more had gravitated to the bottom of Douglas's pouch. No evident source of the pus could be discovered, but it is probable that it came from peritonitis due to over-distension of the bowel. There was no evidence of any strangulation of the hernial protrusion. The small intestine was only moderately distended, but it was crammed with pultaceous, yellow, fæcal contents, and the coats were somewhat thickened. Following it downwards, the distension continued till two feet from the cæcum, where a piece of the bowel passed through the left obturator foramen. Below this the intestine was very contracted. The aperture in the obturator foramen was not large, but the intestine did not appear to be nipped in any way. A knuckle of bowel was in the sac, not quite the whole circumference of it, for a probe could be passed along the intestine across the neck of the sac, but the passage onward would not allow of the introduction of the little finger. The bowel was intimately adherent to the sac throughout, so that on opening the latter the bowel was wounded. The included bowel was greyish, but neither gangrenous nor inflamed. The mesentery was somewhat thickened at its neck, and within the bowel was some old ulceration, as judged from a certain amount of thickening of the edges of an ulcer found at the neck of the hernia. The ulceration was not, however, within the neck, but rather on the opposite unincluded surface of the intestine. The sac pushed the obturator nerve and vessels, with the exception of one branch of artery which passed to the thigh on the inner side, well to the outer side and to its upper part. The obturator muscle was in front of the sac, and necessitated scraping it away to get at it. The sac was of nodular shape about $\frac{2}{3}$ inch in diameter. It caused no fulness externally in the thigh. This was looked for particularly, because the protrusion was first discovered from the inside. In addition to the hernia another coil of bowel (small intestine considerably higher up) was adherent by a strong band at the hernial neck, and about this the distended coils had twisted in a peculiar and indescribable manner, but no obstruction had resulted therefrom, the distension continuing both above and below it. A slight femoral protrusion also existed on both sides. On the right side a little omentum was adherent at the neck of the sac, and by its adhesion dragged down the pyloric orifice of the stomach and the textures in the portal fissure.

The obturator arteries came off from the internal iliacs, and the epigastric vessels were quite normal.

A small stellate ulcer was found on the posterior wall of the lesser curvature of the stomach near the cardia.

The specimen was brought before the Society as a very rare instance of chronic obstruction of the intestine due to obturator hernia. It is, however, not so much a case of chronic obstruction as of obturator hernia, with two distinct attacks of obstruction separated by an interval of some months of health.

I thought that in this it was probably unique, but on looking over the specimens of obturator hernia to be found in Guy's Hospital Museum, four in number including the present specimen, I find one which possibly bears an interpretation of the same kind.

It is stated in the catalogue 2503[80] that a lady, æt. 36, had constipation for some days in September, 1847, and subsequently pains in the abdomen till January 23th, when she was seized with vomiting. The abdomen was opened by Mr. Hilton and the hernia found and reduced, but the patient rapidly sank. (The case is also recorded by Mr. Cooper Forster in the 'Guy's Hospital Reports,' 1864, p. 147 in connection with a second case of obturator hernia of peculiar character, but which does not bear upon the present case. The other two cases, as well as one in the Hunterian Museum, have a history of sudden onset.)

Mr. Birkett also, in his article on hernia in Holmes's 'System,' gives other cases which show that the symptoms of obturator hernia are not always sudden in their onset or rapid in their course. This must depend I suppose upon the completeness or incompleteness of inclusion of the whole circumference of the bowel. In the case recorded here a small channel along which a probe or catheter might pass still existed at the neck of the sac, on the abdominal side of the constriction. One would hardly have thought that this would have prevented symptoms of complete obstruction, yet it appears to have done so. She had, however, lived sparingly and was much emaciated, both most favourable conditions for warding off obstruction to the latest possible period.

Of much surgical interest is the fact that the bowel was so adherent to the sac that even with the parts all exposed and the muscles cut away from the front, yet the intestine was opened in incising

the sac, and I doubt if it would have been possible to return the bowel had any operation been attempted.

Lastly, I may remark that the patient was both old and very emaciated, so that it may almost be said that the probable, and therefore proper diagnosis to have made in her case, would have been that of some cancerous structure.

But though obturator hernia is so uncommon, when it has occurred, it has not unfrequently been associated with those very two conditions which predispose one so strongly towards the diagnosis of cancer, and which have led to the one being mistaken for the other. Therefore, it may be well to bear in mind that the symptoms of obturator hernia may be those of chronic obstruction associated with emaciation, and that it is liable to occur in people advanced in life. It would appear, too, that the hernia being one of the small intestine, the abdomen is less likely to become distended than it is in cancer of the large intestine. *April 8th*, 1876.

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12. *Perforating ulcers of small intestine from a case of strangulated hernia.*

By W. Morrant Baker.

The patient from whom the specimen was obtained was a man, æt. 39, who was admitted into St. Bartholomew's Hospital in February, 1876, under the care of Dr. Southey.

The abdomen was distended, and exceedingly painful and tender. The breathing was thoracic, and the patient had the usual general symptoms of peritonitis.

The patient was taken ill five days before admission into the hospital with severe pain in the left hypochondrium, which was soon followed by frequent vomiting; the pain and sickness continuing with slight intervals up to the date of his admission. The bowels acted last four days before admission, after an enema.

On examination, a femoral hernia, irreducible and with no impulse on coughing, was found on the right side, and a small easily re-

ducible hernia on the left. The patient said he had been ruptured for twenty years; but he thought little or nothing of the matter at any time, and did not think his present illness was connected with the rupture.

It was subsequently discovered that he had been long subject to chronic diarrhœa, with occasional abdominal pain; but he was otherwise a healthy man.

On the discovery of the strangulated hernia the patient was transferred to the care of the surgeon on duty, and in the absence of Mr. Callender, I operated. On opening the sac I found a considerable quantity of omentum and a small strangulated knuckle of intestine at the femoral ring. The intestine which was deeply congested, was easily reduced. Indeed, the stretching of the ring with the finger in feeling for the stricture was sufficient to permit the return of the bowel without any division by the knife.

Part of the omentum was cut off, and the remainder, being adherent, was left in the sac.

On recovering from the anæsthetic the patient seemed relieved, and for the next twenty-four hours he had but little pain, and there was no return of the sickness. Soon after this, however, the pain and vomiting returned, and he began rapidly to sink, and died about thirty-six hours after admission into the hospital.

The *post-mortem* examination was made by Mr. Butlin, to whom I am indebted for the following note:

"The intestines were found everywhere firmly matted together, and on the right side of the abdominal cavity there was a quantity of yellow fluid fæcal matter extravasated.

"The portion of intestine which had been strangulated (about 2 inches of the ileum, a foot above the ileo-cæcal valve) lay just behind the right femoral ring. It was dark from congestion, and its coats were thickened, but there was no indication of gangrene or of ulceration in it.

"About 2 feet above this, in the course of the bowel, in the part of the intestinal wall furthest from the mesenteric attachment, were three or four small gangrenous spots of irregular shape, running transversely across the gut, and surrounded by a small area of congestion. These sloughs extended completely through the coats of the bowel; but only the largest of them had given way, so as to

permit fæcal extravasation. There was some small pitted ulceration in the neighbourhood.

"About a foot and a half above these were several more sloughing spots of smaller size, situate in various parts with reference to the mesenteric attachment. One or two of these did not extend through the bowel, but were limited to the mucous membrane.

"Peyer's patches were all quite healthy; and there was not much indication of enteritis. The large intestine contained dark brown scybala. No obstruction was found at any part. All the other organs were normal."

The pathological interest of the case depends on the nature of the ulceration which caused the patient's death. The ulcers are evidently neither typhoid nor tubercular, nor, in the usually accepted sense, dysenteric. Enteritis, followed by ulcers, not unlike those exhibited, is certainly the result, not very rarely, of intestinal obstruction from various causes; and these ulcers may have been the direct result of the strangulation. But, were this the case, they should be, one would suppose, much more frequently observed in cases of death from strangulated hernia. If, on the other hand, the ulcers were unconnected with the hernia, it is not easy to explain their origin.

Possibly the ulcers were unconnected with the hernia in the first place, but the strangulation of the latter may have made all the difference between a comparatively mild disease and a fatal one. There may have been, that is to say, no perforation, had it not been for the strangulated hernia. It is possible that the supposed rarity of what may be called simple ulcers of the small intestine may be due to the fact that, not very serious in themselves, they are disclosed to view only when by some accident (in this case a strangulated hernia) they prove fatal, or when by chance, during their presence, death occurs from some other cause.

April 18*th*, 1876.

13. *Multiple ulcers of the stomach.*

By W. S. Greenfield, M.D.

Ulcers of the stomach are of so common occurrence that some apology may be needed for bringing this case before the Society. But, although two or even three ulcers may be present in a large number of cases, so large a number as five is comparatively rare. Dr. Brinton states[1] that out of 537 recorded cases, two or more ulcers existed in 114 cases, but in only three cases where the exact number was stated did five exist. The case from which the specimen was taken presented some other features of interest, but need only be detailed so far as it bears on the condition of the stomach. The patient died from perityphlitis and consequent peritonitis.

Elizabeth C—, a widow, æt. 44, was admitted to St. Thomas's Hospital, under the care of Dr. Stone, on January 4th, 1875, suffering from hæmatemesis. She stated that she had had a similar attack of vomiting of blood fourteen years before, but no exact notes of that attack were obtained. She had had repeated vomiting of blood for ten weeks previous to her admission, and on one occasion brought up a pint of blood. This was rapidly relieved by tannic acid and did not recur. There is in the notes of the case no history of any symptoms of ulcer between the attacks. The fatal issue occurred on January 24th, and the *post-mortem* was made nineteen hours after death.

The *stomach* contained a quantity of half-digested egg and milk. At about the junction of the middle with the outer third (*i. e.* cardiac end) of the organ was a constriction about 1 inch in width, producing an "hour-glass" condition. On laying open the viscus its walls were found to be considerably thickened at this point, and only a narrow channel of communication remained between the cardiac and pyloric portions. Immediately to the cardiac side of the constriction, on the posterior wall, was a rather deep oval ulcer, measuring $\frac{7}{8}$ths of an inch in its longest diameter which was transverse to the axis of the stomach, and $\frac{5}{8}$ths of an inch in width. It lay close to the lesser curvature, and was about 1 line in depth. On the pyloric side of the constriction

[1] On 'Ulcer of the Stomach,' p. 12, London, 1857.

were two smaller circular ulcers lying close together, each about ½ an inch in width, and both seated on the posterior wall, and close to the contracted portion. A little beyond them were two other ulcers, one apparently completely healed, represented by a circular smooth patch of mucous membrane deprived of glands, and another a cicatrix only.

The walls of the organ were thickened and infiltrated at the seat of contraction, and the mucous membrane corrugated, but no new growth could be discovered on microscopic examination. The mucous membrane was generally pale, and there was an entire absence of hæmorrhage and of evidence of recent inflammatory reaction about the ulcers.

As a whole the stomach was not enlarged, and there was no marked dilatation of the cardiac portion.

The conditions here found do not appear to throw any light on the mode of production of gastric ulcer. Had the majority of the ulcers been on the cardiac side of the constriction it might have been supposed that their occurrence was favoured by retention. It is not, however, contended that the constriction exerted no influence in inducing them, for no doubt its result would be to check the normal movements, and to permit of that continued contact of the gastric fluid with the mucous membrane, which appears to be one of the most important factors in the production of gastric ulcer. It may be added that this case does not bear out the hypothesis of Dr. Brinton, that where multiple ulcers exist they may be formed by partial healing of a single ulcer in such a manner as to cause a division into two. It would rather appear that the ulcers were of different date, and perhaps commenced at long intervals of time. *May 2nd*, 1876.

14. *Perforating ulcers of the stomach.*

By A. Cummings Air for Wm. Gilson Bott.

H. M. B—, æt. 36, married, had three children, aged 12 and 6 years, and 18 months.

Has suffered from dyspepsia with pain for six years, with occasional periods of complete cessation of all symptoms for two or three months at a time. In May, 1872, received a severe shock, caused by the death of her daughter, who was run over in the street, from which time the symptoms have been more or less constant.

Has never had sickness except during pregnancy. Solid food at all times seemed to cause her less discomfort than fluids, except milk, which was well borne. Broths and soups could not be tolerated. Has always suffered from constipation, and habitually taken aperients.

About two months ago symptoms increased in severity, and about three weeks since sickness commenced "directly after food," and recurred at intervals of two or three days.

I first saw the patient on the 12th of April. She was complaining of persistent pain in epigastric and left hypochondriac regions, sour eructations and constipation. She continued much in the same condition, going about her household duties as usual, until the morning of the 27th, when I was called to her house. I found her standing by the bed, suffering from pain over the whole abdomen, and quite unable to sit or lie down. Her face was pale, pinched, and covered with cold perspiration. Pulse 70, very feeble. I gave an anodyne draught, after which she was much relieved, and remained in this condition until between four and five the following morning when she expressed a desire to relieve the bowels, and in attempting to raise herself to do so, she fell back with a loud exclamation expressive of pain, and died in a few minutes.

Post-mortem ten hours after death.

Body fairly well nourished, of a dusky yellowish hue, and abdomen much distended. On opening abdominal cavity air and yellowish serum gushed out to a considerable extent, the fluid being mixed with particles of green matter, coagulated milk, and oil-globules. The intestines were slightly congested in patches, with a considerable amount of recent lymph deposited upon them and the various

folds of omentum. The colon was very much distended. The stomach was almost empty, and two perforating ulcers, about the size of a florin, were found immediately opposed to each other, near the œsophageal end of the lesser curvature, each ulcer having considerable thickening around it. The pancreas was firmly adherent to the posterior surface of the stomach.

All the other organs were healthy, except the pancreas, which seemed somewhat hardened; a small portion of it will be found attached to the specimen.

I may add the patient's father died a few years ago, having suffered many years from the same symptoms. *May 2nd*, 1876.

(B.) DISEASES OF THE LIVER, PERITONEUM, ETC.

15. *Hydatid cyst of the liver which burst into the lung.*

By W. Cayley, M.D.

Geo. L—, æt. 32, was sent into the London Fever Hospital on August 21st, 1875, as a case of typhoid, of which, however, he presented no symptoms except increase of temperature and slight looseness of the bowls.

He had been unwell for seven or eight weeks, but did not take to his bed till seven days before admission. His symptoms were cough, shortness of breath, and a few days before he had spat a little blood.

On admission the patient was emaciated, and had a phthisical aspect, he suffered from cough, dyspnœa, and expectorated a little tenacious mucus. Pulse 104; temperature 101·4. Tongue was coated with a brown fur, bowels were loose, but the motions presented nothing indicative of typhoid.

On physical examination absolute dulness was found over the back of the right lung below the level of the third rib; over the dull space the breath sounds were absent, and the vocal vibration much diminished. In front the percussion and breathing were normal. The line of dulness in the axilla was almost perpendicular and was not altered

by change of position. The edge of the liver could be felt two inches below the costal cartilages; this was attributed to the organ being forced downwards by the pleuritic effusion, but the patient stated that when seventeen or eighteen years of age he had been treated for enlargement of the liver which lasted two years, and then subsided, and that since he has felt no inconvenience from it.

I regarded the case as one of circumscribed empyema but as the aspirator happened to be out of order, and there was nothing apparently urgent about the patient's condition, I determined to defer the operation of paracentesis for a few days.

On August 25th, while preparations for performing paracentesis were being made the patient suddenly coughed up some small hydatid vesicles, and shortly after expectorated about six fluid ounces of purulent biliary fluid mixed with hydatids. Mr. Murphy, the Resident Medical Officer, now introduced a trocar and canula into the chest in the sixth intercostal space just in front of the border of the trapezius and drew off with the aspirator upwards of two pints of a yellow grumous fluid containing large quantities of hydatid vesicles. The canula was left in. No alteration in the area of dulness was produced; no air entered the chest. Evening temperature 100°; pulse, 152.

26th.—Morning temperature 98·4°; pulse, 140.

The patient passed a good night, but is still coughing up hydatid vesicles. As it was evident that the cyst was only imperfectly emptied, I was anxious to have a counter opening made. Mr. Murphy accordingly made an opening below the tenth rib, three inches behind the first one, and introduced a trocar, but, though water injected through this flowed out through the first opening, no hydatids or yellow fluid could be withdrawn. The second opening was accordingly closed, and the cyst washed out through the first opening by means of the aspiratory syringe, and in this way a large quantity of thick yellow fluid and hydatid vesicles were withdrawn. A drainage tube was then introduced. Evening temperature, 97°. Pulse, 160.

27th.—The patient continued to expectorate hydatids, and the physical signs remained the same. In the afternoon Mr. Murphy introduced a trocar between the seventh and eighth ribs in a line with the posterior border of the axilla and a large quantity of yellow fluid and hydatid vesicles were removed by the aspirator; and by means of these two openings a stream of water was passed through the cyst. A short time after this operation, the patient again began

to cough up hydatid vesicles which poured out of his mouth in a stream, and then fell back suffocated.

On *post-mortem* examination, a cyst with thick fibrous walls, the size of a full grown fœtal head was found arising from the right lobe of the liver; it occupied the lower part of the thoracic cavity and was firmly adherent to the under surface of the diaphragm, to the upper surface of which the collapsed lower lobe of the lung was also adherent. In the lower lobe of the lung was an irregular cavity with gangrenous walls,which communicated with the cyst by a large rugged perforation. Branches of the right bronchus opened into the cavity. The trachea and bronchi on both sides were completely filled with gelatinous hydatid vesicles. The cyst was partially collapsed but still contained a mass of hydatid vesicles and yellow grumous fluid enough to fill a quart measure.

The vesicles when floated in water were found to vary in size from a pea to a large walnut. The yellow fluid gave a well-marked reaction of bile pigment with nitric acid.

The other organs were normal. *October 19th*, 1875.

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16. *On a case of tubercle of the pancreas.*

By Thomas Barlow, M.D.

The existence of tubercle of the pancreas has been either denied or ignored by English pathologists.

Wilks and Moxon say they have never seen an instance, and they consider that the pancreas and salivary glands enjoy an immunity from this form of disease.

In the last edition of Jones and Sieveking there is no allusion made to it.

Amongst the Germans Rokitansky and Virchow say that miliary tubercle is not to be found in this organ.

Förster, however, says that in a few cases tubercle and tubercular cavities have been found in the pancreas.

I have been favoured by Mr. Parker with a quotation from

Friedreich's article on diseases of the pancreas in Ziemssen's 'Cyclopædia.'

"Tubercle of the pancreas is very rare and is found generally as an aggregation of miliary granulations in the form of a more or less cheesy spot, together with chronic tuberculosis of the lungs and intestine." "That which is generally described as tubercle seems to be rather of the nature of chronic cheesy inflammation." "As to the participation of the pancreas in general acute miliary tuberculosis Klebs denies it."

In opposition to this statement, I believe that in the case I have to record, there *was* participation of the pancreas in acute miliary tuberculosis.

H. B—, a little girl, æt. one year and eleven months, was admitted into the hospital for sick children, Great Ormond Street, under Dr. Gee, February 7th, 1875. I have to thank Dr. Gee for permission to bring the case before this Society. The mother gave the following statements. There was a strong history of consumption on both sides. The child herself had never been right since she had had measles twelve months before. There had been discharge from both ears and lumps in the neck, and for more than six months diarrhœa with straining and offensive light coloured stools. She had had a cough for a fortnight.

She was intensely emaciated and semi-collapsed when admitted, and as she died in a quarter of an hour, there was only time to ascertain that she had general impairment of resonance over both fronts, and that some large glands were to be felt in the right iliac fossa.

At the *post-mortem* there was found abundance of transparent and semi-transparent granulations disseminated throughout the lungs but no intervening consolidation. Only one of the bronchial glands was caseous.

In both kidneys there was miliary tubercle in the cortical and pyramidal portions. In the pelvis of one kidney there was a grey granulation, also in its ureter; and on the inner surface of the bladder there were three grey granulations.

On the peritoneum corresponding with numerous intestinal ulcers there were groups of small opaque white caseous nodules, which nodules were continued in the mesentery in linear chains.

The mesenteric glands were caseous and so were the retro-peritoneal.

There were two or three caseous glands close to the head of the pancreas but they were easily separated from it.

The ductus communis choledochus was quite natural.

The exterior of the pancreas showed nothing abnormal to naked eye inspection. It was only on section through the head of the gland that it was felt and seen to be a little firmer and paler than usual. Also on the cut surface of this part of the gland were to be seen about a dozen pale white nodules, each about the size of a small pin's head. They resembled in appearance exactly what one sees sometimes in tubercle of the kidney. These nodules were confined to the head of the pancreas.

On microscopic examination I found numerous mole-hill like aggregations of small celled growth becoming granular in the centre. These aggregations were situated, for the most part, in spaces between the lobules, but they also invaded the lobules and seemed in some spots to have almost replaced the glandular tissue.

In addition to the above aggregations, there was abundance of small celled growth spreading in between the lobules, and especially round the vessels. With a high power many of these small cells were seen to be spindle-shaped with a tail at each end, and there was some fibroid tissue.

All the new growth stained to a less degree than the proper gland elements. As to the gland elements in the above region they were very granular in character.

The above features are brought out in the drawings which Dr. Coupland has made for me from the sections now under the microscope. *November 16th*, 1875.

17. *Enormous dilatation of the bile ducts from stricture of the ductus communis choledochus.*

By John H. Morgan.

The liver exhibited showed a condition of extreme distension and dilatation of the gall-bladder, its duct, and the bile ducts in the substance of the liver.

It was removed from the body of a patient who came to Saint George's Hospital as an in-patient on the 21st of April, 1875, under Dr. Dickinson, to whom I am indebted for the following notes.

His age was 52, and he was by occupation a plumber. He came of healthy parents, and had enjoyed good health till four months before admission, when he had suffered from an abscess in the hand following a prick when at work. This was followed by cold, shivering, and diarrhœa, and a day or two after this jaundice had commenced.

He appeared to be well nourished when admitted; his skin was a bright yellow colour, the irides and conjunctivæ deeply bile-stained. He complained of constant headache and occasional pain at a spot to the right side of the ensiform cartilage. This pain was increased by pressure and aggravated by coughing. The right rectus abdominis muscle was very tense. The tongue was coated and the bowels loose. The fæces were light and stone-coloured, and the urine contained large quantities of bile. No enlargement of the liver could be detected at this time.

In spite of all treatment his condition continued much the same for a month, when he began to suffer from intolerable itching, preventing sleep, and causing him to scratch his skin till it bled. No blood was at any time observed in the motions, and no gallstones were passed.

On July 2nd the liver was found to be much enlarged, both lobes projecting forwards, and giving rise to two tumours, slightly elevated above the surrounding surface of the body. The patient gradually got worse, and died on July 9th.

At the *post-mortem* examination made by myself fourteen hours after death the skin and all the organs of the body were found to be

deeply stained with bile. There was extensive fatty degeneration of the muscular tissue of the heart.

The liver was greatly enlarged and distended; its surface smooth and presenting several slight elevations, whose thin and transparent walls and fluid contents had all the appearance, at first sight, of cysts. These were found especially on the under surface of the left lobe, their sizes varying from an inch and a quarter to a quarter of an inch in circumference. The gall-bladder was very much distended. It measured from five to six inches, and projected some distance below the edge of the liver; it contained dark green inspissated bile mixed with mucus and epithelium. A stricture of the common choledic duct existed just below the point of its formation by the cystic and hepatic ducts. There was but little thickening of the walls of the duct, and below the point of stricture it was patulous and of the normal colour and calibre; above it was dilated to the size of a large finger, and stained green with bile. On the outer side of the duct the duodenum was found to be bound down to it by adherent bands of lymph, and on tearing them apart a perforating ulcer was found to exist at this spot in the walls of the gut, which was only prevented from extravasating its contents by this adhesion. The contraction of this lymph round the duct had caused its constriction. Another ulcer not so far advanced was seen to exist close to this one.

The result of this constriction was an obstruction to the onflow of the secretion, and hence a dilatation of all the ducts. This had caused the distension of the gall-bladder and its duct, and also those of the liver, which it had distended to such an extent as to cause their extremities to project on the surface as the cysts above described. The circumference of the duct of the left lobe measured over an inch. These cysts collapsed on pressing out their contents, which consisted of viscid, slightly green mucus, and epithelium.

A very similar condition of the ducts of the liver was exhibited before this Society in the year 1860, by Mr. Holmes, in which case the stricture was supposed to be the result of the passage of gall-stones. It was published in the 'Transactions' of the Society, and the specimen is now in the Museum of St. George's Hospital.

The preparation shows the liver and its ducts, but the amount of dilatation cannot be correctly estimated from the present condition of the ducts. I have kept them as far as possible distended with horsehair, and the larger ones are laid open to show their size. The

pyloric end of the stomach, the opening of the common duct into the duodenum, and the two ulcers, one perforating the other not, are also shown. *December 7th*, 1875.

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18. *Congenital deficiency of the common bile duct, the cystic and hepatic ducts ending in a blind sac; cirrhosis of the liver.*

By J. Wickham Legg, M.D.

A poor woman brought her baby, a little girl, to me, at St. Bartholomew's Hospital, on June 24th, 1875. The child was then seven weeks old. The mother said it was jaundiced at birth, and that the tint since birth was becoming deeper. The eyes at birth were yellow. The motions were said to be quite white, like curds and whey, and the water was said to be yellow. After birth the child is said to have had, for three weeks, a breaking out on the skin "more like small pox with yellow heads." There was no bleeding from the navel after birth. This is the seventh child; none other has had jaundice or bleeding from the navel. The six children are alive and well. She has never had any miscarriages.

The child is now universally jaundiced, with small hæmorrhages like flea-bites all over the arms. The child snuffles, but has no rash now on skin, or sores about vagina or fundament. The child coughs, and all over chest may be heard sibilant rhonchi. The liver is not to be felt. The amount of dulness seems natural. Ordered to take Vin. Ipecac. ♏j, Am. Carb. gr. ss, syrup ʒij, every four hours.

June 28th.—The cough is gone, and there are now no signs of catarrh. The jaundice is no less. The motions continue white. The water cannot be saved, but the napkins are stained yellow.

July 1st.—The jaundice is thought to be deeper. There is no pain on handling the belly. The liver cannot be felt; its dulness begins two fingers' breadth below nipple, and stretches four fingers' breadth below, that is to one finger's breadth below margin of ribs. The baby is said to be a "good baby and to give no trouble;" it takes food well, and sleeps well at night.

19th.—Has not been to the hospital since July 5th. There is no change in the belly or size of liver. The child is wasting. There has been a rash of pruriginous strophulus over the child for the last three days. There are no hæmorrhages; the jaundice is thought to be deeper.

July 22nd.—The child seems somewhat less yellow. While straining at stool, blood came along with motion. Child is said to be always asleep.

29th.—The jaundice is thought to be less. Blood is now passed with every motion; colour of stools no longer white, but obscured by the blood, which is said to be great in quantity.

August 6th.—The liver may now be easily felt. It has a sharp edge, and feels hard. The dulness begins two fingers' breadth below nipple, and stretches to two fingers' breadth below ribs. No blood now passed with motions. To take Pot. Iod. gr. j in water three times a day.

September 3rd.—The mother says the child is better. She has noticed a tinge of yellow in the motions. For the last three weeks it has taken one grain of carbonate of ammonia in syrup three times a day.

10th.—The mother says the child is not so well. There have been "little white blisters" in the mouth, which have been rubbed with borax and honey. None was seen at the time of the visit. There is no intense yellowness. The liver extends to three fingers' breadth below margin of ribs. The spleen is likewise much enlarged, and lies in the left lumbar region, very freely moveable. There is no ascites, but the child has piles. The napkins are seen to be still coloured yellow by the urine, and the fæces are white.

24th.—The mother came to say that the child had died at four o'clock that morning. For three days before death it was said to have had repeated attacks of "convulsions."

Examination thirty-three hours after death.—Body weighs 3400 grammes, universally jaundiced, and wasted. No fat apparent anywhere.

Nothing unnatural in brain or membranes. Fontanelles widely open.

No fluid in peritonæum or pleuræ. The pericardium and heart natural.

The upper lobes of both lungs natural; the lower lobes are solid, plump, sinking in water, not depressed below surface of pleura, nor

is pleura wrinkled. The cut surface is smooth and shining, and no fluid escapes on pressure.

The intestines contain colourless fluid, no solid fæces. They are natural in appearance.

The round ligament of liver is natural. The portal vein and hepatic artery appear quite natural, and free from clots. The gall-bladder is shrunken, holds a small amount of a yellow fluid, and the cystic duct opens without any winding into a cyst, the size of a largeish marble, placed to the right side of the portal fissure, between the liver and duodenum in the hepato-duodenal ligament. This cyst likewise receives the hepatic duct coming from the liver. It is a blind sac, and no passage can be found into the duodenum. The cyst is attached to the duodenum by a broad thin membrane, in which no duct can be found. The cyst is lined by a membrane about a millimeter thick, showing numerous small vessels, but otherwise closely resembling an hydatid cyst; it peels off with very little trouble from the outer wall of the cyst. It lines the whole of the cyst, except where the cystic and hepatic ducts enter, and here it is perforated by two round holes. The cyst holds a yellowish fluid. On attempting to dissect the hepatic duct it is found free, and holding a yellowish fluid as far as a few lines from the liver. Here the walls of the duct become very thick and fibrous, the open mouths of a few narrow ducts being seen to open into it. On dissecting beyond this fibrous band into the right lobe, the duct from the right lobe is found to be greatly dilated, and to hold a quantity of thick bile; after being thus dilated for half an inch, however, it quickly becomes of small size, and cannot be followed with a fine pair of scissors. The duct from the left lobe cannot be followed at all.

The liver, stomach, pancreas, and duodenum weigh together 270 grammes. The liver is of a deep olive-green colour, the surface granular, and marked with numerous branching white lines. The largest of these white lines correspond to depressions. On section, the cut surface is seen to be of the same colour as the peritoneal surface, but studded with islets of white tissue, which apparently correspond to portal canals. In other places the islets of tissue appear green, surrounded by the white tissue. There seems to be no dilatation of the hepatic ducts, and bile does not flow out of cut surface. The liver is very hard, both to feel and knife.

The spleen is large, long, and narrow, and weighs 90 grammes. On section it is not harder or softer than natural; the Malpighian

bodies are very large, but not easily distinguishable in colour from the pulp.

The stomach and duodenum are natural. There is a papilla where the bile duct ought to open. Pancreas natural. Kidneys natural.

The membrane lining the cyst into which the gall ducts entered was looked at through the microscope the day of the examination after death. It showed a distinctly fibrous structure. The fibres were in some places coarse, in others fine; between them were many granules, just large enough to be seen, but at times so big as to show a double outline. In the midst were seen many irregular granular bodies of a high red-brown colour, without any distinct arrangement, form, or size. They seem to be made up solely of granules.

Parts of the liver were hardened in very weak chromic acid and the sections made were stained in carmine and mounted in glycerine. With the low power the section shows very distinctly dark green islets of liver cells imbedded in broad bands of connective tissue. The liver islets themselves are full of dark green pigment balls, which sometimes look as if in the cells, sometimes as if between them. With higher powers (Hartnack Oc. 3, Obj. 9. *à im.*) the liver cells are seen to be not much changed in shape or size. They have dark highly granular contents, but a well-shaped nucleus. Between them are dark green masses of pigment of various shapes, chiefly cylindrical, sometimes with two branches; in other places they have been cut directly across and show a round section. They are probably casts of the intercellular bile-ducts. The broad bands of new connective tissue are made up chiefly of fibres having a wavy course and perforated by many rounded holes apparently for vessels. The tissue is remarkably devoid of nuclei or lymphatic bodies. This is the more noteworthy because in all other morbid growths of connective tissue within the liver these bodies are very prominent; as in cirrhosis. The connective tissue penetrates within the lobules, and in many places a fibrous network can be seen where the liver cells have been displaced.

These cases of congenital deficiency of the gall ducts are very rare, not more than twenty being on record, two of these being in the 'Transactions' of this Society.[1] It is of some importance to

[1] The following are the cases to which I refer:
Anderson, 'Boston Med. and Surg. Journal,' 1850. Vol. xli, p. 440.
Binz, 'Arch. f. path. Anat.,' 1866. Bd. xxxv, p. 360.

inquire in what relation these cases of jaundice stand to those other cases of jaundice, which are still rare, but yet more common than these, which are accompanied by bleeding from the navel and sometimes by signs of a more wide-spread hæmorrhagic disposition. Some circumstances are common to both. Both seem to seize upon boys more commonly than girls; both show the same disposition to appear in children born of the same parents. In both the jaundice appears within a few days after birth. Here, however, the likeness stops. For in those accompanied by hæmorrhage death commonly puts an end to the disease soon after the bleeding has first appeared, while those who are only jaundiced may live for weeks and even for months. There are about 220 cases on record of bleeding from the navel, and of these 84 have been jaundiced;[1] but careful records of the state of the bile-ducts have been given in but very few cases; not more than seven or eight, if so many. Of these the bile-ducts were absent, thickened, narrow and free from bile, or plugged, in four. It is indeed unfortunate that in a disease which proves so often fatal, and which must therefore have offered so many opportunities for examining the bile-ducts, only seven reports of their state out of 220 cases should have come down to us. Most of these cases of hæmorrhage from the navel have been seen in America. Taking all the facts, I think it will be most in accordance with present knowledge to class together both these sets of jaundice, with or

Campbell, 'Northern Journal of Medicine,' 1844. Vol. i, p. 237.

Donop, 'De Ictero, speciatim neonatorum,' Diss. Inaug. Berol. 1828. p. 20.

Freund, 'Jahrb. f. Kinderheilkunde,' 1875. Bd. ix, p 178.

Hennig, ibid. p. 406.

Heschl, 'Wiener med. Wochenschrift,' 1865. p. 493.

Home, Sir Everard, 'Phil. Trans.,' 1813. Part II, p. 156.

Köstlin, 'Württemb. med. Corresp.-Bl.,' 1862. Nr. 14; also in 'Canstatt's Jahresb. f. 1862.' Bd. iii, p. 293.

Murchison, 'Clinical Lectures on Diseases of the Liver,' London, 1868. p. 363.

Nunneley, 'Trans.' of this Society, 1872. Vol. xxiii, p. 152.

Romberg and Henoch, 'Klin. Wahrnehm. u. Beob.,' Berlin, 1851. p. 158.

M. Roth, 'Arch. f. path. Anat.,' 1868. Bd. xliii, p. 296.

Virchow, 'Ges. Abh.,' p. 858.

C. West, 'Lectures on the Diseases of Infancy and Childhood,' London, 1874, p. 623.

Wilks, 'Trans.' of this Society, 1862. Vol. xiii, p. 119.

[1] Grandidier, 'Die freiwilligen Nabelblutungen der Neugebornen,' Cassel, 1871.

without umbilical hæmorrhage, as one kind. Dr. West has adopted this theory without discussion.[1]

One point in the history of these cases must be early discussed, and that is their relation to syphilis. What evidence is there in this particular case of the presence of syphilis? The child was the last of seven, all the rest being alive and well. The woman had had no miscarriage. After death no changes which could be laid down to syphilis were found save only the disease of the liver and ducts. On the other hand, it may be said that the child had the snuffles, but I think this may, without wresting the facts, be set down to the general catarrhal state and not necessarily be of specific origin. Of the nature of the skin diseases which troubled the child for the first three weeks of its life I can give no opinion, the disease having disappeared when the child was brought under my care. In one of Binz's cases the father had some years before marriage suffered from a sore on the penis followed by bubo. Binz, therefore, thinks it possible that syphilis may have had some share in the matter, since perihepatitis is so common in syphilitic infants. He, however, remarks that no other signs of syphilis were present. M. Roth looks upon his case as one of syphilis because perihepatitis is so commonly seen in congenital syphilis; there were also present "cold" abscesses of the scalp and neck, and fatty degeneration of the neuroglia cells. Whether this author looks upon the constant appearance of the compound granulation corpuscles in the brain of infants as morbid I do not know. There was no exanthema, œdema, or other scars present. Of the other cases nothing is said about syphilis. It thus becomes apparent that it cannot be accepted as a general proposition that syphilis is present in all these cases, for in one only is there any evidence, and that by no means conclusive, of its existence, setting aside the lesions in the liver.

The length of time which this child lived was, according to the mother, five months and a fortnight. She would not or could not tell me the day of its birth. It is one of the longest on record, Campbell's case having lived six months, and Dr. Nunneley's and Heschl's seven months each.

There is no case precisely like the present on record. That of Anderson's approaches most nearly to it. At the junction of the

[1] West, 'Lectures on the Diseases of Infancy and Childhood,' London, 1874, p. 624.

cystic and hepatic duct there was a slight prolongation answering to the first beginnings of the common duct, but which ended in a blind sac. What in this case is so noteworthy is the dilatation of the blind end of the cystic and hepatic ducts into so large a cyst. This seems best accounted for on the supposition that the duodenal end of the common duct was either never developed or else very early obliterated, and that the other end of the duct, continuing to receive the bile, became dilated into a cyst. The nature of the membrane lining the cyst is more obscure. It is clearly not an hydatid, which it resembled very closely to the naked eye, as the membrane was vascular and showed none of the characteristics of an hydatid under the microscope. The appearances seen with the microscope suggested rather an old blood clot, though as to the way in which the blood became extravasated into the cyst and formed so equal a lining, I am unable to set forth any theory.

It is probable from a comparison of the cases on record, that an overgrowth of the connective tissue of the liver or a cirrhosis accompanies all these cases of congenital deficiency of the bile-duct. Which is the primary disorder, the disease of the bile-ducts or of the liver? I should be inclined to answer, of the bile ducts. For cirrhosis, as a primary disease, is exceedingly rare in infants, even if they be syphilitic; and it is unlikely, if the disease descended from the liver, that it would involve the bile-ducts alone, and leave untouched the other vessels in the portal canals.[1] Again, it is likely beforehand that obliteration of the ducts would beget an overgrowth of the connective tissue in the liver, as it has been shown by experiments on animals that the ligature of the common duct causes in the same way overgrowth of the connective tissue.[2] The presence of a constriction of the hepatic duct just at its entrance into the substance of the liver would favour, on the other hand, the belief that the obliteration of the ducts was due to a perihepatitis, and it is noteworthy that the ducts within the liver were not dilated

[1] It would have added greatly to the value of F. Weber's case of congenital jaundice and cirrhosis ('Beiträge zur pathologischen Anatomie der Neugebornen,' III Lief., Kiel, 1854, p. 49) if some account of the state of the gall-ducts had been given. Cf. a case of cirrhosis and jaundice at p. 45.

[2] Wickham Legg, 'St. Bartholomew's Hospital Reports,' 1873, vol. ix, p. 161. At the end of my paper (p. 181) I have drawn a comparison between the artificial ligature of the ducts and their congenital obliteration.

but reduced to fibrous chords, just as in Cheyne's case of hæmorrhage from the umbilicus and jaundice.[1]

I am inclined to think that in the present case the ducts were deficient early in fœtal life; at all events they were obstructed before birth, as the child was born jaundiced. In some cases, however, it is said that no jaundice was noticed at the time of birth, but that it came on some days later. This may be due simply to a want of taking notice in the mother and nurse, as what is more likely than that a slight jaundiced tint should be missed by those unaccustomed to look for it? This is made more likely by a case of Dr. Wilks, in which the mother said that no jaundice had been noticed for fourteen days after birth, and yet the child had never passed any meconium, the stools being always white. It seems highly improbable that so complete an obstruction of the ducts as is implied in white stools should exist for fourteen days without early begetting jaundice. Still cases do exist in which the jaundice is said to have set in after birth, and in which the first stools have been of a dark green colour—a fact which seems proof of the permeability of the ducts a very short time before birth.

It has seemed to me that some of the cases of jaundice followed by umbilical hæmorrhage might be explained on some such theory as the following. Immediately after birth the large vessels on the under surface of the liver, the ductus venosus and the branch to the portal vein, begin to be obliterated. In nearly all children this process runs quite a natural course. But in some the process of obliteration spreads too widely and extends into the porta of the liver along the connective tissue which wraps round the portal vein and the gall-ducts. One result of this is an obliteration of the gall-ducts, or at least a decrease in the bore of the ducts, so that jaundice is set up. This again will be followed by an overgrowth of the capsule of Glisson, aided further by an extension of the same process that first acted on the gall-ducts to the portal canals. This being the case, the portal circulation can no longer be easily carried on; there is an impediment to its passing through the liver, and the blood, therefore, passing in the direction of the least resistance would enter the branch of the left portal vein and again press up the umbilical vein and ductus venosus. The blood would thus find an easy exit from the vessels of the navel not yet perfectly healed.

[1] Cheyne, 'Essays on the Diseases of Children,' Edinburgh, 1801. Essay II, p. 9. Note.

This theory of of a portal congestion would likewise explain the fact that bleedings into the portal tract, into the stomach and intestines, are so much more common in these cases of umbilical hæmorrhages than hæmorrhages elsewhere. The spleen is also of large size.

The facts in favour of this view are few in number, and, indeed, the theory is suggested rather for the purpose of awakening the attention of morbid anatomists to the vessels on the under surface of the liver than with any belief in its being found adequate to explain all the phænomena of the disease nor every case. In most cases of umbilical hæmorrhage the fœtal passages have been found open, whereas they should be closed by the fourth or fifth day. This is certainly a most serious objection to any theory starting on the ground of increased activity of the obliterating process. Then as to the state of the portal vein, in this case it was natural. In one of Binz's cases it was obstructed. In another the right branch is said to have been filled with clots. In a third the portal vein is said to have been patent. Thus if we have little information about the state of the gall-ducts, we have less concerning the blood-vessels.

It is noteworthy that in some of these cases the umbilical vein is said to have contained "pus." Thus in Herapath's case the child was seized with jaundice and erysipelas of the hand a few days after birth. There was a slight hæmorrhage from the umbilicus. After death pus was found about the joints and also in the umbilical vein and portal vein within the liver. The gall ducts were impervious.[1]

Deeember 7th, 1875.

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19. *Cirrhosis of the liver in a child aged ten years.*

By T. Henry Green, M.D., for T. D. Griffiths, M.D., of Swansea.

This specimen of cirrhosis of the liver is from a female child ten years of age, who died on November 17th.

Family history.—The parents are respectable tradespeople; both healthy; married fourteen years ago, and have had seven children. There

[1] Herapath, 'Med. Times and Gaz.,' 1854. Vol. i, p. 286.

is no suspicion of syphilis in either. A most careful inquiry was made on this point in the father's case. The mother first of all miscarried at three months; in the next place she gave birth to a fine healthy child, which died, according to her account, of "inflammation of the brain" while teething, at the age of from ten to twelve months. The child was ill for a few days only. The second child also died about ten months old from "the effects of measles and bronchitis." The third child was delicate, "had inflammation of the lungs and bronchitis, and at last died of consumption," about the age of two years and a half. The patient was the fourth child. The youngest three children are living and fairly healthy. The patient had all the ordinary infantile diseases, viz. whooping-cough when she was about a year old, scarlet fever at three and a half, measles and chicken-pox at six, and mumps at seven years of age. She also suffered from infantile paralysis for three or four weeks when about two years of age. Early in 1870 she suffered very severely from sickness for about a week or more, and then became jaundiced for about a fortnight. After this attack the little patient enjoyed good health until the latter end of 1871, when her appetite failed, and she had another mild attack of jaundice, which lasted about three or four weeks. Before the jaundice had entirely disappeared my friend Dr. F. T. Roberts, of University College Hospital, saw the patient with me. The liver was then, as it had been for some weeks, greatly and uniformly enlarged, extending as low as the level of the umbilicus, the surface was smooth, the margin hard and well-defined, not tender on pressure; in short, the organ appeared to have all the physical signs of a large lardaceous liver. The heart and lungs were considered healthy; there was no albumen in the urine, no ascites, or anasarca. The little patient had never suffered from rickets, diseased bones, or a chronic abscess. There was no enlargement of lymphatic glands or any indication of anæmia; she never suffered from any affection of the eyes; *never took alcoholic drink* [a most careful and searching inquiry was made on this point], or made use of any irritating food more than children generally of her age. There was no history of tuberculisation or of pleurisy. The object I had in showing the case to Dr. Frederick Roberts was to point out the difficulty felt in coming to a conclusion as to the cause and real nature of the enlargement of the liver. The history of the case excluded the probability of the enlargement being tubercular, leukæmic, hydatid, fatty, or lardaceous. We were also satisfied that the enlargement was not due to cancer or simple congestion, and thus were left for the time in the dark. Early in 1872,

when every trace of the jaundice had disappeared, the liver remained *in statu quo* as to size and hardness. The child had now recovered her appetite, and was improving in health. As a matter of curiosity I called to see the child in the early part of the summer of 1872; she appeared in fairly good health, but on examination the liver was found to have diminished but little in size. From that time until the latter end of last September the little patient enjoyed good health; she was slim and tall for her age, very intelligent, and quick at her lessons; face oval; teeth regular, well shaped, but rather large, and ivory-white in colour. Her appetite was always good, and her spirits were buoyant; she never missed a day's schooling through ill-health. Towards the end of September last the mother sought medical advice for a swelling in the child's belly, which she declared had come on in the course of a few days.

Now the case had assumed a very different aspect. The hepatic dulness was nil in the mammary line when the patient lay on her back; the spleen was greatly enlarged; the peritoneal cavity was distended by fluid; no anasarca in the lower limbs.

In the course of a fortnight the skin assumed a yellowish tint, and in another week the child was decidedly jaundiced; the ascites was on the increase, and the superficial veins on the abdomen and front of the chest were well marked. The jaundice had disappeared entirely by October 25th, when paracentesis was first resorted to, to relieve the distressing symptoms which threatened life. The operation was performed the second time, on November 15th, and two days after the patient died, aged 10.

On *post-mortem* examination the liver was found to be an excellent specimen of a small contracted (cirrhosed) liver [the liver, heart, and kidneys were exhibited at the Pathological Society]. It weighed only 15 ounces; the peritoneal covering was smooth and shiny all over; the whole of the organ was hard, very tough, and irregularly lobulated, and granular on the surface. On section it appeared to the naked eye to consist chiefly of tough fibrous tissue interspersed with irregular lobules (varying in size and colour) of the true liver substance, in different stages of strangulation and degeneration. The portal veins were greatly enlarged. The spleen was as large as the liver, hard and tough.

Both lungs were healthy; there were no pleural adhesions. There was a slight cloudy swelling at the edge of the mitral valve—of the parts corresponding to the attachments of the chordæ tendineæ.

Remarks.—This is an excellent specimen of a cirrhosed liver from a child aged 10, without any evidence whatever, *on the most careful and satisfactory inquiry*, of it having been produced by the use of alcoholic drink or irritating food of any kind; neither was there a particle of evidence of hereditary syphilis, of previous history of rickets, diseased bones, or chronic abscess.

December 7th, 1875.

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20. *Cancer of the pancreas and liver; cancerous polypi of the portal vein and pancreatic duct.*

By J. Wickham Legg, M.D.

Susannah K—, aged 78, was admitted into St. Bartholomew's Hospital, under the care of Dr. Brunton, on August 10th, 1875.

She said that she had enjoyed very good health until about a year ago; she had never been jaundiced; she had suffered from vomiting in the morning for nearly a year. For the last three months the vomiting had been constant; for the last few days the legs and ankles have swollen.

The woman is very thin. The belly is much distended. The liver is greatly enlarged, reaching to the right iliac region, to two inches below umbilicus, and to within two inches of left anterior superior iliac spine. Large nodules may be felt all over the liver surface. The heart appears natural.

She continued to vomit, and no relief to this symptom could be secured. On October 5th it is noted that the urine is acid and contains a trace of albumen. She died on October 28th at nine in the morning.

Examination twenty-seven hours after death.—Body greatly wasted. The liver and nodules on it can be very easily felt. The liver itself seems smaller than during life.

The peritonæum holds a very considerable excess of a clear fluid.

There is some excess of fluid in both pleuræ. The pleura of the apex

of the right lung is much thickened, and below it are many highly pigmented nodules; the same on the left side. The upper lobes of both lungs œdematous, less so the lower lobes. No new growths in the lungs.

Pericardium.—The vessels at the base of the heart are bound together by many old and œdematous adhesions; over the cardiac layer of the pericardium are many white spots. The heart after being opened weighs 250 grm. The coronary arteries are very tortuous; the heart itself wasted. Both the tricuspid and mitral valves show a very great thickening and opacity, not merely of the edges, but of the whole of the valves. The small flap of the mitral is likewise very much larger than is usual. The sigmoid valves are natural beyond some slight thickening.

The large and small intestines and mesentery appear quite natural. The spleen weighs 80 grm.; it is very small; it has numerous thickenings of its capsule, very like articular cartilage in physical character.

The porta of the liver shows several enlarged lymphatic glands. On cutting them across the section shows a white soft surface, with many small hæmorrhages. The common duct is greatly dilated; on dividing it the flat surface is three times as broad as it should be. This dilatation persists until half an inch from the opening into the duodenum, where it rapidly becomes of a natural size. The duct is everywhere stained yellow. The hepatic duct is likewise much dilated, more on the left than the right lobe. The cystic duct has lost its windings, and opens at once into the gall-bladder. The gall-bladder is very small, not more than an inch long and a quarter of an inch across; it is still stained yellow; there are no gall stones in it.

The portal vein outside the liver, the splenic, and superior mesenteric veins are natural and free from clots. The hepatic artery is natural.

The liver itself weighs 2500 grm. On the surface are many nodules, about twenty, of size varying from a mustard seed to a small apple. Their outline is rounded, colour whitish, and some are distinctly umbilicated. On cutting into the liver, however, the nodules are far more numerous within. They are round, white, rather firm, and many show small hæmorrhages. The liver substance between the nodules is natural, save that the centre of the lobule seems darker than natural. The hepatic veins are natural. On dissecting the portal vein within the liver, one of the first divisions of the right branch is found filled by

several polypoid bodies. They are smooth and club-shaped at the end, where they project into the free cavity of the vein; above they become slightly adherent to the vein-wall, which appears perfectly natural. They send prolongations up branches of the portal vein above them, where after travelling up some short distance they again end in a bulbous projection. Their origin, however, appears to be in each case in one of the large nodules of the right lobe, to which they are attached by long and narrow pedicles. None of these polypoid bodies can be found in the left lobe; they are of about the same consistence as the nodules themselves.

A large lymphatic gland, similar in appearance to those in the porta, is attached to the under surface of the pancreas, but does not enter its substance. The pancreatic duct where it makes the turn round the head of the pancreas is for about an inch filled by a long polypoid body, very soft, which is adherent only at the end near the head of the pancreas. The body of the pancreas seems natural. The head is enlarged, and the cut surface shows many yellow cheesy spots, from a mustard to hemp seed in size. The head is rather softer than the body.

The stomach and duodenum natural; no thickening of pylorus.

Many prævertebral glands are enlarged, and like those in the porta of the liver, one especially between the vena cava and aorta, as low down as the fork.

The kidneys weigh 180 grm. The capsule tears off with some trouble, leaving a red granular surface; the cortex much narrowed and indistinct in structure. In the pelvis of one kidney is abundance of red yellow gravel.

The bladder and rectum natural. The ovaries show small cysts, none bigger than small marbles. Around the right is a small extravasation of blood. The uterus small, puriform fluid in cavity.

The aorta is dilated and atheromatous, so are all the large vessels. The vena cava free from thrombus.

The head not opened.

The polypoid bodies from the portal vein were examined by means of Hartnack's microscope the same day (Oc. 3, Obj. 9, *à im.*). The field was flooded with oval nuclei; many large cells, containing abundance of fat, and with these nuclei in them, were likewise seen. The nuclei often contained two or three nucleoli. Acetic acid brought out the nuclei well, leaving the fat drops undisturbed.

A scraping from the head of the pancreas taken from a place close

to the root of the polypus examined: an immense number of compound granulation corpuscles were seen, and a large number of cells closely resembling those from the polypoid bodies in the portal vein. A creamy fluid exuding from the root of the polypus showed the same appearances.

The tumours being hardened in chromic acid were examined with the microscope in the month of December. Thin sections stained with carmine were mounted in glycerine, and looked at with Hartnack's microscope, Oc. 3. Obj. 9. *à im.*

The polypi in the portal vein showed a delicate network supporting cells. The walls of the alveoli were very thin, while the meshes themselves were wide and rounded. The walls were formed of an almost homogeneous material, scarcely fibrous, and showing very few nuclei. The cells were all contained in the meshes of the network: they were somewhat tightly packed, inclining to be polyhedral in shape, containing a large nucleus about the size of a white corpuscle; sometimes two nuclei were present in one cell. The contents of the cells were highly fatty and granular. Precisely the same appearances were found in the tumours in the liver.

The polypus in the pancreatic duct showed appearances of the same kind, only the walls of the alveoli were thicker and more fibrous, and the spaces between them narrower. The cells, too, seemed more tightly packed and more abundant, for they only slightly exceeded in size the nuclei, which appeared in many places, when the section was thick enough, to touch each other. The nuclei were large and stained deeply with carmine. In the head of the pancreas the meshes were much narrower, and the walls of the alveoli very thick and fibrous. The cells show the same characters as above.

A case like this must, I think, be clearly separated from those which have been recorded as cancer of the portal vein. In such cases the portal vein, both within and without the liver, is reported to be filled with a thick, creamy matter, which is assumed to be of a cancerous nature. I do not think there is any good evidence in these cases that this matter is really cancer. Of course, before the microscope came into such general use as at present such appearances in the portal vein were at once set down as cancer. But even in those more lately published, such as a most carefully worked-up case of Spaeth,[1] the his-

[1] Spaeth, 'Arch. f. path. Anat.,' 1866, Bd. xxxv, p. 432. This writer gives a very complete and accurate account of the bibliography up to his time. For that

tological evidence of a cancerous growth in the vein seems weak. It is based solely on the presence of large cells in the material filling the vein—an opinion in which the author is clearly influenced by the now extinct views of Lebert as to the cancer cell. This and the rest of the cases of cancer of the portal vein on record which I have looked through cause me to entertain grave doubt if their exact nature have been really made out. It is impossible to avoid drawing a comparison between them and cases of suppurative thrombosis of the portal vein. Paulicki's case is, perhaps, better established, but even that shows no convincing evidence.[1]

The only cases that I have been able to find which closely resemble this are described by Carswell, who gives a drawing of a polypus hanging from a cancerous nodule into the portal vein,[2] and by Frerichs, who speaks of five such cases coming under his observation. His description of the naked-eye appearances exactly corresponds to this specimen. Frerichs thinks that in these cases the cancer first attacks the wall of the vein, and thence spreads into the free bore of the vessel. The wall of the vessel becomes thick, and sends forth new growths into the vein.[3] This case has not furnished me with any means of verifying or disproving this statement.

In reference to these polypi I have only further to add that I scarcely think they are so rare as the few cases on record would lead us to believe. The branches of the portal vein are but rarely dissected out, and unless this be done it is, of course, unlikely that such would be noticed. For the same reason it likewise seems possible that the polypus in the pancreatic duct may not be a very rare appearance, seeing how unusual it is to look through the pancreatic duct in its length. *January* 18*th*, 1876.

before 1843 the reader may consult Puchelt, 'Das Venensystem,' Leipzig, 1844, Theil ii, p. 297.

[1] Paulicki, 'Berlin klin. Wochenschrift,' 1867. p. 393

[2] Carswell, 'Illustrations of the Elementary Forms of Disease,' London, 1838. 'Carcinoma,' plate iv of first series, fig. 4.

[3] Frerichs, 'Klinik d. Leberkrankheiten,' Braunschweig, 1861, Bd. ii, p. 278.

21. *Cirrhosis of the liver in a child aged six years.*

By W. Cayley, M.D.

Ada F—, æt. 6 years, was admitted into the North Eastern Hospital for Children on June 23rd, 1875.

The mother has had five children; the eldest died of bronchitis at the age of two years, the second is living and healthy, the third is the present case, the two youngest are living and well. All the children had snuffles and thrush when infants, but quickly recovered; they did not waste, and had no cutaneous eruption, or bad eyes, or sores of any kind. The mother presents no trace of syphilis. She denies ever having given her children wine or spirits. Ada sometimes had a little beer for dinner, but did not care about it, and the mother discouraged her from taking it, as she did not "hold with giving children intoxicating liquors."

The child is stated to have had no illnesses since whooping-cough at the age of nine months. About six days before her admission the mother noticed that her belly began to swell.

On admission the child was pale and sallow. Conjunctivæ slightly yellow; did not complain of pain. The belly was greatly distended, with a distinct sense of fluctuation; circumference 27½ inches. Superficial veins on the right side enlarged. Legs œdematous. Occasional cough; a few dry râles were audible over the chest. Heart sounds normal. Urine sp. gr. 1020, free from albumen, not very highly coloured.

June 25th.—Paracentesis was performed, and 47 ounces of clear serum drawn off by the aspirator. The belly rapidly filled again, and on June 29th the operation was repeated, and 37 ounces of a similar fluid were withdrawn.

July 9th.—The child was tapped again, and 42 ounces were withdrawn. An obstinate diarrhœa now set in, attended by bloody motions, and the fluid did not again collect in very large quantities, but the child became greatly debilitated and emaciated.

On July 26th epistaxis occurred, and still further reduced her strength. An irregular nodular mass could now be felt in the epigas-

trium, which appeared to be formed by the left lobe of the liver. The lower border of the right lobe of the liver did not extend below the cartilages. The diarrhœa was checked by opiates and astringents, and she regained flesh and strength, and on September 30th was sent to our Convalescent Home at Croydon, where she remained six weeks and then returned home much improved.

She continued pretty well till December 23rd, when she was suddenly attacked in the night by hæmatemesis, and vomited about a pint of blood. The next day she was brought again to the hospital and admitted. She was much blanched and very feeble.

On December 26th and 28th she had attacks of epistaxis. Some general anasarca now showed itself, but no marked ascites.

On January 4th she was again attacked by hæmatemesis to a slight degree, and the following day convulsions supervened, and she died.

When in the hospital the first time she was treated with iodide of potassium and iron.

On *post-mortem* examination the liver was found to be pale, tough, and granular. Its capsule was not thickened, and there were no cicatricial depressions or gummata. On microscopical examination it presented well-marked cirrhotic characters, the organ being intersected by tracts of lymphoid and spindle-shaped cells, intermixed with a certain quantity of fibrillated connective tissue. These tracts separated irregular groups of liver-cells. Weight of liver, 17 ounces.

The mucous membrane of the stomach and intestines was pale, and showed no traces of the hæmorrhage which had taken place during life.

The kidneys were pale and anæmic, the surface smooth.

Mr. Needham made a careful microscopical examination of the other organs, and furnished me with the following report:—

"Analogous changes to those in the liver may be observed in the other organs examined, viz. the heart, kidneys, spleen, stomach, and brain. The walls of the blood-vessels in all the organs appear thicker than natural, and some of the vessels can be easily mapped out by the number of small round cells, like those already described, surrounding them. The amount of this infiltration varies considerably in the organs referred to, being most distinct in the stomach, and least so in the heart muscle. In addition to these changes the connective tissue is increased in each organ, and the proportion of fusiform cells is greatly in advance of the normal. The epithelia of the uriniferous

tubules is very granular, and the tubes themselves in many places are filled with granular matter.

"The gastric follicles are greatly obscured by the immense numbers of the small round cells which have infiltrated the mucous and submucous layers. The solitary follicles are unusually distinct.

"The structure of the spleen also appears greatly altered by the presence of these small cells; in many places little else can be seen but these elements enclosed by the bands of the fibrous trabeculæ.

"Between the muscular fibres of the heart many of the small round and an increased number of fusiform cells may be observed."

January 18*th*, 1876.

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22. *Tubercular phthisis; ulceration of larynx; extreme tubercular disease of liver; large soft spleen; tubercle in the kidneys.*

By James F. Goodhart, M.D.

William V—, æt. about 35, was admitted under the care of Dr. Pye-Smith on December 13th, 1875. He had cough and hæmoptysis for about six months, and hoarseness had been present for some five weeks. The right chest was flattened, with deficient expansion and dulness; loud cavernous respiration was heard above the right clavicle; and below, bronchial breathing, with much coarse crepitation. The laryngoscope showed much thickening of the epiglottis.

The patient was very pale and had much epistaxis.

Autopsy.—Nourishment spare. Brain, normal.

The right pleura was covered with recent lymph, and the cavity contained a quantity of fluid; the left side was healthy.

The right lung had a cavity of small size at the apex, and the whole lung was studded with aggregated clusters of yellowish tubercles.

In the left lung there was much scattered caseating tubercle in the upper lobe, spreading down the front of it by preference; the base was extensively consolidated by recent pneumonia, which had evidently

started in isolated lobules. It did not show any tendency yet to become caseous.

Larynx.—Both sides were ulcerated. The epiglottis was much thickened and ulcerated. The mediastinal glands were not caseous, but they were a little enlarged.

Liver.—Very large; colour dark; surface studded all over with yellowish-white patches, which at first sight were suggestive of pyæmia or cancer; the anterior edge was thick, and projected some way beyond the ribs. Taking the organ in the hand, it was felt to be decidedly nodular or tuberculated, and on close inspection the nodules were seen to be made of aggregated tubercles. These gave to the section of the liver a very remarkable appearance such as I have not seen before. The surface was a little uneven from the slight projection of the clusters above the surrounding level; it was mottled all over, which might, perhaps, best be described as a large-type nutmeg condition. The tubercular patches were yellowish, and the intervening liver substance was of a dark purple from extensive congestion. The patches varied much in size, were very irregular in outline, infiltrating the liver rather than growing as separate nodules; at any rate there was nothing like a capsule at any part; they were very numerous through the whole liver. In many cases they spread from the coat of the hepatic vein into the surrounding liver tissue, and showed no preference, as is usual, for the portal channels or Glisson's capsule.

The gall-bladder was distended with black bile. Portal fissure normal.

Supra-renal capsules normal.

Large pulpy spleen, but without tubercle, or at most a few on the capsule.

The kidneys both contained several tubercles, in the substance and on the surface.

The blood was examined, and at most only contained a slight excess of white corpuscles.

Under the microscope the clustering of the tubercles around the hepatic venules was not so evident; indeed, the greater number were located, as usual, in the portal fields, though not exclusively confined to them. In the more diseased parts the liver structure was hardly recognisable; but in its place was a vascular connective tissue crowded with nuclei. The early stages presented very similar appearances to those of early cirrhosis of the liver or of the indurated liver of heart disease, but yet they were peculiar; so regular in the arrange-

ment of the nuclei and stroma in their relation to each other, that I am inclined to think the growth started in the nuclei of the capillaries, or, at any rate, that changes occurred *within* the lobules simultaneously with those *between* them, and, therefore, that no process of infiltration was in course of progress.

I was further disposed to think that the nuclei of the hepatic cells multiplied and helped to form the new growth. Whether this was really so, or whether the hepatic cells were only undergoing an atrophic process due to pressure, dependent upon a germination of the nuclei of the capillary plexus around them, is a question it is hard to settle beyond the reach of criticism. But if the former be the correct interpretation of the appearances, the case would seem to belong to the group of malignant tumours, even though it be tubercular. For it is not uncommon to find in such, when they occur in the liver, a general overgrowth of intercellular fibrous tissue, as well as the distinctive cellular one ; that is to say, a mixed process of overgrowth of pre-existing tissue and new growth. And it has also been shown, particularly by Dr. Creighton, to be characteristic of malignant tumours in the liver that the hepatic cells themselves proliferate and take part in the formation of the disease. Neither of these features is a common one in tubercle of the liver as usually found, one only in cirrhosis and syphilis. But both are present in Hodgkin's disease. The structure of the nodules may, therefore, not unfairly be called lymphomatous, and the case one of lymphoma of the liver, associated with tubercle of other organs. *January 18th*, 1876.

Report by the Committee on Morbid Growths on Dr. Goodhart's case of extreme tubercular disease of the liver in a case of phthisis.—On microscopical examination it appears that there is considerable change, even in those parts of the liver which to the naked eye appeared normal. The regular arrangement of the cell-columns is interfered with ; considerable tracts of them are flattened against one another, exactly as is seen in the neighbourhood of cancerous nodules ; yet there is nowhere any distinct growth independent of the pre-existing tissue, and pushing it aside.

In almost all other parts of the organ the spaces between the hepatic cells are enlarged, and nuclei or cells are found in them. Sometimes these are collected in pairs, as if they had been produced by fission ; and an obvious suggestion is that made in the above report, that they may have arisen from a proliferation of the capillary nuclei. When the

change has reached an advanced stage these cells are present in large numbers; they are embedded in a stroma of indefinite character. Here the original hepatic tissue is in a state of atrophy. The hepatic cells are small and collected together in clumps, which, containing several nuclei, give rise to an appearance similar to what would be observed if these cells were themselves proliferating.

There is also a considerable amount of cell-growth in the fibrous tissue, which lies between the lobules, and the outer coat of the hepatic vein is also in some places crowded with cells.

After consideration we think it safer, as an equally good interpretation of the appearances we have described, and perhaps on the whole more probable, to call the case one of extreme overgrowth of tubercle in the liver, rather than to adopt unreservedly the other explanation which has been favoured by one of us as the exhibitor.

C. Hilton Fagge,
James F. Goodhart.

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23. *Typical cirrhosis of liver in a boy aged nine.*

By C. Murchison, M.D.

Henry N—, æt. 9, schoolboy, admitted into St. Thomas's Hospital September 5th, 1875. Father alive and in good health; mother died of phthisis, and several brothers and sisters had died in childhood.

As far as boy was aware, he had always had good health until about two months before admission. During whole of July he had suffered from sickness and vomiting every morning. On August 1st he had been sent to sea-side, but the sickness persisted and abdomen began to swell. After sixteen days he returned home, and the sickness now ceased, but the swelling increased. From first he had been losing flesh and bowels had been rather confined.

After boy's death it was ascertained that his father kept a low public-house, and that the boy had been in habit of drinking a good deal of wine and water, especially between meals. While in hospital also he took stimulants with a readiness quite unusual in children.

On admission boy was emaciated, but abdomen was very large owing to fluid in peritoneum. Girth at umbilicus 32½ inches; abdomen not tender; lower margin of liver could not be felt; upper margin not too high; spleen much enlarged; lower end fully 4 inches beyond ribs; no jaundice; no œdema of legs; no albuminuria; no sign of constitutional syphilis and sounds of heart normal. Temperature occasionally as high as 100·6°. Eats and sleeps well. Tongue normal.

Boy was at first treated with syrup of iodide of iron internally, while tincture of iodine was painted over abdomen. On September 8th and 9th temperature in evening rose to 102·8° and 103·4°, but usually it was under 100°. On September 18th girth at umbilicus had increased to 34¼ inches. Citrate of ammonia was now substituted for the iron, and a mercurial plaster was applied to abdomen, while bowels were kept open; under this ascites rapidly diminished, and on October 5th no trace of it remained. Girth at umbilicus was only 24 inches, and except that spleen remained large and he was weak, boy seemed well. He was now treated with iodide of potassium and iron, digitalis and cod oil. Once or twice he was sick in morning; and on October 24th abdomen seemed to be swelling again, girth being 25½ inches. After this swelling rapidly increased, until on November 10th girth was again 34½ inches, and tongue red and dry; bile in urine. Digitalis and iodide of potassium with aperients produced no effect; and on November 12th girth 37½ inches; integuments of abdomen were smooth and shining; pulse 120; respiration 60, embarrassed. Fifteen pints of clear straw-coloured serum were drawn off by paracentesis, and patient was ordered blue pill, squill, digitalis, and saline diuretics. The fluid rapidly reaccumulated, and on November 24th girth 38 inches, pain in abdomen, and occasional vomiting; considerable œdema of legs; extreme dyspnœa and prostration. Paracentesis again performed, and sixteen pints of fluid drawn off with temporary relief; but next day patient complained of intense pain in abdomen and nausea, and abdomen was refilling. At 7 p.m. collapse came on, and at 11.20 p.m. he died.

Autopsy.—Peritoneum contained 6⅓ pints of ascitic fluid, containing a few flakes of lymph. Peritoneum generally much injected. Great omentum matted into a mass adherent to adjacent intestines. Mesentery thick and œdematous. No tubercle. Liver small; weighed 21½ ounces, the normal weight for patient's age being about 32 ounces; outer surface presented typical hob-nailed character of cirrhosis; numerous small irregular yellowish prominences, separated by pinkish-grey depressions; substance very hard, tough, and leathery.

On section yellowish islets of secreting tissue, surrounded by fibrous bands; gall-bladder contained normal bile. Spleen large, weighed 10 ounces, deep red, firm. Mucous membrane of stomach thickened, deeply injected, and with much adherent viscid mucus. Lower part of ileum congested. Peyer's patches and solitary glands of large intestine slightly enlarged. Kidneys large and congested, but structure normal. Heart and lungs normal, with exception of some hypostatic congestion of both lungs and small ecchymoses in subpleural tissue.

Remarks.—The main interest of this case consists in its being an example of true cirrhosis of the liver in a boy aged 9. Rare cases of cirrhosis of the liver in children have been recorded by different observers, and have been frequently referred to as proofs that true cirrhosis does not necessarily result from the use of alcoholic or other stimulants. But in many of these cases the apparent exceptions, on careful investigation, have confirmed the rule. Niemeyer, for example, quotes two typical cases of the disease observed by Wunderlich in two sisters, aged eleven and twelve years; on careful inquiry it was found that both of them had been great dram-drinkers.[1] Not long ago Dr. Wilks had a little girl, eight years old, under his care in Guy's Hospital, "suffering from what proved to be a very small hob-nailed liver; she had been addicted to drink, having taken as much as half a pint of gin daily."[2] It must be remembered also that in some of these cases it is very difficult to arrive at the truth respecting the patient's previous habits. Since the case which I have now related occurred I have had under my care a girl, aged 19, suffering from cirrhosis and ascites, who, though a barmaid, declared that she had drunk positively nothing in the way of wine or spirits; but on inquiry it turned out that her statement was quite untrue. So far as my experience goes, I have never met with a typical example of hob-nailed liver (as distinguished from other forms of contracted liver) after death, at any age, in which there could be made out a clear history of moderation in the use of alcohol. In early life it is not improbable that the activity of the liver may render it more liable to suffer from alcohol and other irritating ingesta, than in adults. *May 16th*, 1876.

[1] 'Practice of Medicine,' American edition, vol. ii, p. 641.

[2] Dr. Moxon, 'Guy's Hosp. Reports,' 1875.

24. *Receding gummata of liver in case of congenital syphilis.*

By Thomas Barlow, M.D.

R. H—, a male child, aged twelve weeks, was brought as an out-patient to Great Ormond Street, on the 28th of April, 1876.

There was a history of sores on the tongue (of which the scars remained) in the mother, and of premature and stillborn children.

This child was full time. Nothing wrong was noticed till he was seven weeks old; then he began to snuffle and his skin to peel.

When he was brought, at twelve weeks old, to the out-patient room he was still snuffling a little. There was a slight scaly eruption on the face, the belly, and the buttocks, and $\frac{1}{4}$ inch from the anus, on each side, were two oval symmetrical sores, not condylomata, but superficial ulcers.

He was ordered mercurial ointment inunction into the soles.

In a week's time the snuffling was better, the sores on the nates were improving, desquamation was less, but some fissuring at the corners of the mouth had become manifest.

At the lower end of the right ulna there was some swelling of the epiphysis and the contiguous ends of the first and second phalanges of the left index finger were swollen. There was also what I took to be a "cellular node" over the middle of one ascending ramus of the lower jaw.

The liver did not extend more than two fingers' breadth below the chest-margin, which was not too much for a child of that age. The spleen extended two fingers' breadth below, which, of course, was abnormal, but a common-enough condition, as Dr. Gee first pointed out in the *early* stage of congenital syphilis.

The child was extremely marasmic.

The mother's milk was scanty and poor; in spite of an extra supply and the rubbing-in of cod-liver oil, &c., the child sank on the 13th of May, *i. e.* when fourteen weeks old, and having had mercurial treatment for a fortnight.

At the *post-mortem*, only the viscera were allowed to be examined. With the exception of the spleen and liver the viscera were natural. The spleen was simply enlarged; it was firm on section, and presented no naked-eye alterations.

The liver weighed 8½ oz.; it did not seem enlarged. On the surface were eight depressed areas, varying in size from that of a split pea to that of an almond. One had a slight tail-like prolongation. There were two or three on the under surface. Cutting into these areas, they were seen to define circumscribed thimble-shaped masses, which differed extremely little in colour and consistency from the surrounding liver-substance, only that they *were* circumscribed, and had the faintest dash of buff colour. There was no general induration of the liver, but in two or three places the portal canals seemed thickened. There was one mass at least, entirely *in the substance* of the liver, with similar characters to the above.

Under the microscope, sections of one of these masses showed it to be fairly vascular. There was excess of fibrous tissue round the vessels, and separating and replacing the liver-cells a nuclear growth without any special arrangement.

There was no caseation anywhere to be made out.

I think I am justified in calling these masses, on account of the depression on the surface, *receding* gummata. I venture to suggest that in the form in which they occur in this specimen they are a middle term between the common form of gumma in the liver and the fibroid patches seen sometimes on the surface of adult syphilitic livers. Is the absence of caseation in these growths to be attributed to their vascularity? If so it is a distinction from tubercular growths, and it is a clue to their capability of being absorbed.

The case is interesting clinically because of its supporting one of Mr. Hutchinson's propositions as to the coexistence of gummata with secondary symptoms. Here were what I take to be receding gummata at the time when the squamous eruption was not yet disappeared, when there were symmetrical sores on the nates, and when even the snuffles had only just begun to subside.

May 16th, 1876.

V.—DISEASES, ETC., OF THE GENITO-URINARY ORGANS.

(A) KIDNEYS, BLADDER, ETC.

1. *A case of carcinoma lipomatosum of the kidney.*

By C. Hilton Fagge, M.D.

The preparation was taken from the body of a woman, æt. 67, who died in Guy's Hospital of dropsy, secondary to bronchitis and emphysema. Her urine gave a slight cloud of albumen with heat and with nitric acid. It deposited epithelium and mucus, and a few casts which are described as having been of granular, almost fatty appearance. Its specific gravity was 1022.

The left kidney weighed 4½ ounces; it was very granular on the surface and showed a number of cysts of different sizes. The cortex was reddened, its tissue blurred and diminished in thickness. Its surface was sprinkled with a number of minute yellow grains which, under the microscope, appeared to be masses of some soft fatty substance.

The right kidney weighed 7 ounces. When its capsule was stripped off, its surface appeared comparatively smooth. It showed a few small yellow points like those seen in the opposite kidney. Its surface was not granular, but it showed two or three rounded whitish nodules which were subsequently seen to be connected with the mass of growth presently to be described.

On cutting this kidney open we found that its whole centre was occupied by a soft yellowish substance, looking something like softening decolourised coagulum, and forming large rounded masses. It filled the whole hilus, but it did not lie within the pelvis; this, on the contrary, being compressed by it. It also stretched the calyces and flattened the pyramids. Into the branches of the renal

vein, on the other hand, it grew freely; and it projected as a mass of the size of a pigeon's egg into the interior of the trunk of the vein outside the kidney. Another mass, the size of a walnut, lay in the interior of a branch of vein, which must have been greatly dilated by it, and which seemed to be going to join the main vessel at some little distance from the organ. Even within the kidney a good deal of the mass evidently lay within venous channels expanded to receive it. Some of it, however, grew into the tissue of the cortex, and one or two nodules could even be seen on the surface.

Under the microscope, in the fresh state, the growth showed large fatty globules, so that, as in the case of a very fatty liver, it at first looked as if it were made up only of fat. The fat-globules, however, were really contained in the interior of cells of very irregular forms, with large oval nuclei. Some of the cells were quadrilateral, some pear-shaped, and some had the form of the cells of cylinder epithelium. The tumour was not degenerating, but simply infiltrated with fat. At the growing margins it showed the same peculiarities of structure.

Both kidneys appeared under the microscope to be diseased to about the same extent. The connective tissue between the tubules was greatly thickened; the capsules of the Malpighian tufts were fibrous; the epithelium was very opaque.

After being hardened in chromic acid, a portion of the growth showed the characteristic alveolar structure of a carcinoma. The cells were now observed to have well-formed nuclei, and some of them contained two nuclei or even more. The bright fat-globules were still visible in the cells, and in some parts the fat had crystallised in the usual stellate form. Round the carcinomatous nodules there was a fibrous capsule, in which cell proliferation was apparently going on.

Remarks.—The specimen is one of a very unusual kind, and I do not remember to have ever seen one like it. The only description which I have met with of a growth presenting similar microscopical characters is in the work of MM. Cornil and Ranvier. These writers give among the varieties of carcinoma one which they term "carcinome lipomateux." In it "the cells contained in the alveoli are filled with drops of fat, and may resemble the cells of ordinary adipose tissue; but the tracts of fibrous tissue which support the latter are wanting." MM. Cornil and Ranvier have

twice observed this form of growth, one of their cases being a carcinoma of bone. "Such tumours," they add, "are so like lipomata that they might be mistaken for them if one did not take all their characters fully into consideration."

November 2nd, 1875.

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2. *Displaced right kidney.*

By J. Wickham Legg, M.D.

I have brought this specimen before the Society because I believe displacement of the kidney to be somewhat rare. At the least during the six years that I have been in the *post-mortem* room at Saint Bartholomew's, I only remember to have seen one other case; moveable kidneys, of course, I exclude.

The specimen was taken from the body of a little boy, about three years of age, who died on November 5th, and was examined on November 8th, 1875. The cause of his death was tubercular meningitis. Tubercle was likewise found in the lungs and liver.

On taking out the liver and intestines the right kidney was seen at once to be displaced. It lay at least an inch lower than it should, there being a space of an inch between the kidney and the supra-renal bodies. Both supra-renal bodies were in their right places. The left kidney was in its usual place, and the vessels and ureter quite natural and arranged as is common, save that the ureter was somewhat dilated. The right kidney, of natural size, lay on the psoas muscle, its middle part being on a level with the crest of the ileum. The hilus, by which the vessels enter and escape, was on the fore part of the kidney about its centre. There were two right renal arteries both arising near the same place, about half an inch from the origin of the inferior mesenteric. The smaller passed horizontally into the upper part of the kidney without entering the hilus. The other, passing over the inner part of the kidney, entered the hilus. This artery was accompanied by a renal vein which entered the inferior cava at the level of the artery. Another vein, much larger than this, arose on the outer side of the hilus, passed obliquely over

the surface of the kidney to enter the inferior cava at the level of the left renal vein. This large vein was accompanied by a smaller vein which arose from the upper part of the kidney and which entered the vena cava a little below the larger vein, but nearly at the same place.

It is the rule in cases of displaced kidney that the supra-renal capsule should not descend with the kidney, but stay in its natural place, and it is also the rule for the vessels to arise at a part of the aorta on a level with the displaced kidney. It would also seem to be the custom for the hilus in these cases to be on the fore part of the kidney and not on the inside. It is not, however, the rule for the right kidney to be displaced; it is far more commonly the left. According to Klebs, this is as it should be, and cases of displaced kidney on the right side should be viewed with suspicion, being most probably cases of moveable kidney only.[1] This case, at all events, was not one of acquired moveableness. The kidney was perfectly fixed when first seen to be displaced, and I look upon the origin of the renal artery below the inferior mesenteric as proof of the congenital origin of the displacement.

The specimen is preserved in the Museum of Saint Bartholomew's.

There is likewise another specimen of displaced kidney in the Museum of Saint Bartholomew's put up about three years ago, and of which no account has, I think, been published. The *post-mortem* examination was made by Dr. Gee, and the specimen was presented by him to the Museum. It was taken from the body of a man, aged 24, who died with adherent pleuræ, dilated bronchi, and solid calcareous masses at the apex of the lung. The following appearances about the kidney were found. The supra-renal capsules were in their natural place. The left kidney lay on the spinal column between the common iliac arteries; not easily moveable, that is, not a floating kidney. It was somewhat smaller and rounder in shape than natural. The pelvis lay between the kidney and spine. The arteries were three in number, and supplied from the right common iliac; there was no trace of a left renal artery. The renal vein was about natural, passing upwards to the vena cava inferior. The ureter natural. The pelvis was somewhat distended. The right kidney was natural.

January 4th, 1876.

[1] Klebs, 'Handb. d. path. Anat.,' Berlin, 1870. p. 610.

3. *Bladder and prostate from a boy who had been cut for stone eleven years previously.*

By James F. Goodhart, M.D.

The boy, a shoeblack, fourteen years of age, was cut for stone, so he stated, at three years of age. Since then he has always passed urine involuntarily.

He was attacked with acute rheumatism a fortnight before his admission to Guy's, and died from that disease complicated with extreme myocarditis, pericarditis, and double pleurisy.

The bladder was contracted, but not otherwise abnormal, except that when the parts were removed from the body a very evident white mark extended from the left side of the prostate to within two or three lines of the left ureter, indicating a somewhat extreme incision in the floor of the bladder. But after the specimen had been decolorised by preservation in spirit this was no longer visible, neither was there any corresponding cicatrix, so that it probably corresponded to a mere split in the mucous membrane, and not to any deep incision.

The prostate is much distorted by a large and puckered cicatrix, which occupies its floor. The caput gallinaginis is pushed well to the right of the median line, and only one vas deferens (the right) can be traced onwards to the floor of the urethra. In front of the cicatrix are two deep sacculi, which run in some indurated cicatricial tissue forwards in the floor of the membranous urethra.

The base of the bladder was carefully dissected by my friend Mr. Donbavand. The vesiculæ seminales were normal and the right vas deferens, but the left could not be pursued further than the base of the prostate. Here it entered some dense cicatricial tissue, and nothing could be passed along it. The testes were healthy.

The muscular tissue of the bladder at the neck, as elsewhere, was quite normal microscopically.

As a case showing the state of the bladder and prostate many years after the operation for lithotomy it has its interest, and may be compared with one recorded by my colleague Dr. Fagge in 1875, but the special point raised by it is, "What light does the state of parts throw upon the incontinence of urine which occurs occasionally in children after lithotomy?" From what can be gathered from books

no explanation of such an occurrence has been offered; yet it is by no means uncommon. It has been thought by Mr. Marsh that possibly the state of the muscular tissue or nerves about the neck of the bladde should account for it, but I do not think so; the muscular fibre appears very good; of the nerves it is more difficult to speak, for some fibres may well be involved in the very dense cicatrix in the prostatic region. I could make nothing of them microscopically. Mr. Walsham has suggested to me that the difficulty in such cases is a mechanical one and similar to that met with in some instances of incontinence where the prostate is enlarged, and where, by reason of distortion of the canal, the passage is not completely closed. This is by no means improbable, and the specimen so far supports such a conclusion in that there is much irregularity in the channel in front of and in the prostatic portion, the caput gallinaginis being much twisted to the right side.

April 28th, 1876.

(B) MALE GENITAL ORGANS.

(C) FEMALE GENITAL ORGANS.

4. *Dermoid ovarian cyst.*

By J. KNOWSLEY THORNTON.

S. B—, æt. 59, came into the Samaritan Hospital on January 5th, 1876, under the care of Mr. Spencer Wells. She was seen by Mr. Wells shortly after admission, and the abdominal distension was so great, and the suffering consequently so severe, that Mr. Wells requested me to tap her the same evening.

I removed 86 pints of rather thick fluid looking like pus, and with a faint disagreeable odour. When the cyst was about three quarters emptied masses of white fatty material began to come away, and soon the canula blocked, and on clearing it with a probe an immense quantity of these little round brown bodies came away, and soon the top of the fluid in the footpan was covered with a thick coating of them as they floated to the surface. As will be seen from the specimen I hand round they are about the size of mustard seeds,

some larger, some smaller, of a pale brown colour, with a granular surface, as if made up by the cohesion of a number of still smaller bodies.

An immense quantity of them came away and a good deal more of the white fat, and then the canula blocked again, and on injecting some carbolic lotion to try and clear it, the whole contents of the cyst suddenly became solid, and of the consistence of a bladder of lard. Owing to the enormous abdominal distension the wall of the abdomen was lying over the edge of the bed like a collapsed bladder, and the external cold and the cool lotion caused the fat to become solid.

The application of hot-water bags and injection of hot water at last enabled me to empty the cyst, which was well washed out with large quantities of carbolic lotion.

The patient, who was extremely emaciated and debilitated, was much relieved for a few days; but she then had a severe attack of bronchitis, chiefly, if not entirely, due to the condition of the diaphragm after its long and great distension, and the generally weak state of the patient allowing accumulation of mucus in the lungs, which she had no power to expectorate.

This condition was successfully treated by Dr. Day; but she died, worn out, on the 27th of January, three weeks after the tapping.

The fluid, when allowed to stand quiet for a few hours, separated into three layers; the upper fourth consisted entirely of these little brown bodies; then came a layer of liquid fat, about half the whole volume, the bottom of the glass being occupied by a fluid looking like pus, but found, on microscopic examination, to consist entirely of squamous epithelium in masses and single scales, in every stage of change and degeneration. Mixed with it there were a few short broken red hairs and cholesterine scales.

I placed some of the little brown balls in ether, which dissolved them, or rather separated their component parts, and I found their colour due to short red broken hair and cholesterine scales, the rest of them being epithelium, stuck together with granular fatty matter.

I found that by warming some of the fluid in a test-tube, and slowly and constantly shaking it round and round for some time, I could manufacture some little balls similar to those in the fluid. The smell seemed due to the glandular structures in the cyst-wall,

and was similar to that often observed in sebaceous cysts. I could find no evidence of any putrefactive change.

The *post-mortem* examination made by Dr. Day and myself eighteen hours after death showed that the lungs had almost recovered a healthy condition.

On cutting through the abdominal wall one passed at once into an enormous cavity with smooth walls, and containing a little fluid similar to that described above, and some more of the little balls, also a ball of matted red hair as large as a hen's egg; at the lower part of this cavity was a mass looking very like the uterus and ovaries, uniformly enlarged, but found to be a mass of secondary cysts, one of which was also dermoid, the rest like ordinary ovarian cysts.

The cyst-wall and abdominal parietes were so intimately adherent from the pubes up to the ensiform cartilage it was impossible to separate them, and on cutting through the posterior wall of the cyst the abdominal organs were found crushed upwards and backwards, but none of them adherent to the tumour. The uterus and left ovary were quite free from the tumour, which sprang from the right ovary. The uterus was large and the other ovary small and shrivelled, but with a thin-walled cyst as large as a marble projecting from its surface.

It is worthy of remark that the patient had been once tapped before, nine years back, when exactly the same quantity of fluid was removed, but the cyst not nearly emptied, as the canula became blocked. The tumour was diagnosed after her first confinement, in 1841.

The little brown bodies I show to the Society as pathological curiosities, as I can find no report of anything similar ever having been found or described. The case is also of great interest as showing that a dermoid cyst may attain this enormous size. There was evidently some slight admixture of serous fluid like that in ordinary ovarian cysts, but the main bulk was fat and epithelium.

It would have been quite impossible to separate the cyst from the abdominal wall during life, but I think it is a question worth considering whether in any similar case the dermoid cavity might not be opened and cleared out, a permanent opening being left, or, in other words, the skin lining the cyst being turned into an external skin. In this particular case the mass projecting at the

bottom of the cyst might easily have been ligatured and entirely removed without necessarily opening into the peritoneum. The accompanying figures show some of the many forms of epithelium, and the natural size and appearance of the little balls.

April 4th, 1876.

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5. *Gangrenous ovarian cyst.*

By J. Knowsley Thornton.

Gangrene of an ovarian cyst is fortunately a rare occurrence, more so perhaps than might be expected when we consider the position and connection of such a cyst with a tolerably long pedicle. There are numerous recorded cases in which the pedicle has been twisted and its vessels obliterated, or the whole torn through, so that the tumour received its blood supply through adhesions to neighbouring organs. In such cases the adhesions probably save the patient from the serious risk of gangrene by serving as channels for a new blood supply, and possibly in more cases than we know of adhesions are really useful by regulating the blood supply during chance obstructions or partial obstructions to its flow through the pedicle, as well as by holding the tumour in a certain fixed position, and thus lessening the risk of twisting of the pedicle.

The cyst which I show to the Society to-night I removed by ovariotomy at the Samaritan Hospital, on October 27th, to give the patient, who was "in extremis," a last chance of life, but though she rallied slightly, she only lived sixteen hours.

The following is a brief history of the case:—

S. E—, æt. 28, married, and mother of one child, a girl, æt. 15 months, was placed under the care of Mr. Spencer Wells at the Samaritan Hospital, on October 12th, by Dr. Parsons, of Wimbledon. She was a small, bright, healthy-looking woman, with a fresh colour. On examination she was found to be pregnant, and, according to her own calculations, about 4½ months. The abdomen

was occupied by a good-sized cyst on the right side, and rather behind and to the left the enlarged uterus was found. The cyst was freely moveable, and from the character of the fluctuation seemed to consist chiefly of one large cavity.

Dr. Playfair had diagnosed ovarian tumour at King's College fourteen months before, *i. e.*, just after her confinement. Six months before the birth of her child she had an attack of severe pain, &c., which was probably due to the tumour.

I tapped the patient by Mr. Wells' request on October 19th, and removed ten pints of ovarian fluid, but it was evident there were some masses of smaller cyst left behind. On the 20th she complained of headache, and had a quick pulse; temperature 100·0°; very free perspiration. On the 21st her temperature rose in the evening to 101·2°, but after this she seemed to improve, though still subject to headache and heavy sweating; temperature and pulse, however, not much above normal. I suggested to Mr. Wells the propriety of performing ovariotomy, but he thought it would be best to keep her quiet in bed and watch her, and doubtless she would soon have recovered the effects of the tapping had her condition not led to obstruction to the circulation of the cyst.

October 26th.—I was suddenly called to the patient, and found her complaining of extreme pain in the abdomen, which had come on suddenly after turning rather quickly in bed; pulse rapid and feeble; temperature falling; face dusky. I feared some purulent, possibly putrid fluid, had escaped from the cyst into the peritoneum. She passed a tolerably comfortable night with poultices over abdomen and small repeated doses of Tr. Opii per rectum, but I found her in the morning with temperature 98·0°; pulse 140; face dusky, cold perspiration, and suppression of urine. After consultation with Dr. Savage (Mr. Wells being unfortunately out of town), I told her we thought it advisable to perform ovariotomy at once, and to this she anxiously assented, saying she felt she should die if something was not done. The operation was unavoidably delayed till three in the afternoon. Dr. Godson administered bichloride of methylene, and I then carried her to the lift, and she was cold, blue, and almost pulseless when placed on the table. She rallied slightly during the operation, and for a few hours afterwards was bright and cheerful, and talked to her husband. On opening the abdomen I found the enlarged uterus surrounded by coils of intestine distended by gas, but no tumour was visible. Searching with my hand I found it,

however, but far back and on the left side instead of on the right side. The peritoneum contained a considerable quantity of reddish serum with a mawkish but not putrid odour. The intestines and other organs were congested. The tumour was mottled black and white and dull, and coated with lymph in patches, evidently in a partially gangrenous condition; this being due to the pedicle, which was very short, being twisted three times on itself, and the tumour, which was of the right ovary, having fallen completely over to the left side of and behind the enlarged uterus.

The connection with the uterus on the right side was so close that I was obliged to tie the pedicle in two halves after transfixion, the ligatures being cut off short and left in the peritoneum.

The case raises an interesting point as to the propriety of tapping or performing ovariotomy during pregnancy, as it is plain a similar accident might readily happen again. The facts that the child was certainly alive a few hours before the operation, and that through the whole case ending in a severe operation and death no sign of abortion occurred, seem to me strongly to support the advisability of performing ovariotomy at once, if any operation is necessary—a course also encouraged by Mr. Wells' successful cases.

The tumour contained one large cavity with four and a half pints of ovarian fluid in it, and there were masses of smaller cysts projecting into the main cavity at various points. The large cyst was lined with a thick layer of dark lymph-like material, soft and friable; this readily peeled off, showing an intensely congested granular surface underneath. The looser textures of the cyst wall were black with clotted blood, the firmer fibrous parts were white and dead-looking, but evidently contained some extravasated blood. This tumour I showed to the Society in its fresh state, in which it was evident that its whole substance was infiltrated with blood, parts at any rate having passed from the stage of intense congestion to that of acute gangrene. Careful microscopic examination of sections, made from portions hardened in chromic acid and stained with log-wood, showed the various parts of the tissues broken up and packed in the most remarkable way with red and white blood-cells. The vessels were everywhere much distended with blood clot, and in many places broken, the clot in the vessel being continuous with extensive extravasations among the tissues. In some of the portions examined the individual bundles of connective tissue and cells composing the cyst wall were so thoroughly separated that the tissue

was evidently killed; but in other parts the various stages of clot organisation could be beautifully studied, and the advanced stages seen in some parts showed that though the fatal strangulation probably took place when the severe pain and collapse came on, congestion to an unusual extent had occurred some days before. It seems likely from the symptoms observed after the tapping that some amount of inflammatory action was set up by that operation, that as the tumour became heavier from the increase in the quantity of contained blood congestion was increased, and the enlarged condition of the uterus raising up the cysts caused a drag on the pedicle and obstruction to the flow of blood, which would of course be felt much more by the large thin-walled veins than by the arteries which in the ovary and ovarian tumour have remarkably thick muscular walls. Movements of the fœtus may have aided the twisting of the pedicle. Thus the tumour was already in an inflamed congested state, with its circulation becoming more and more impeded, blood being pumped in through the arteries, but finding little or no escape through the compressed veins. Supposing the patient to have remained quiet on her back, the condition might have become serious, a sudden change of position on to her left side precipitated the threatened evil, and the whole tumour became gangrenous.

Not only were the component elements of the tissues separated by masses of blood-cells, but the small cysts were found entirely filled with blood-clot, and it is worthy of remark that the epithelial lining seemed entirely destroyed both in the large cyst and in the smaller microscopic cysts; indeed, in no portion of the tumour which I examined could I detect any trace of the epithelial structures which usually form such a prominent feature in all ovarian tumours. This fact, together with the processes of organisation of the blood-clot observed in many parts, suggest the possibility that had the entire strangulation not occurred, the changes taking place in the tumour might have ended in its destruction or conversion into a mere mass of cicatricial or fibrous tissue.

While studying the changes to be seen in the blood extravasated among the tissues and in the clots filling up the cavities of the small cysts, I met with an explanation of an appearance which had often puzzled me when examining sections of the walls of ordinary ovarian tumours—an appearance of a very fine lymphadenoid tissue, similar to that forming the central part of ordinary lymphatic

glands. This I had often seen in patches and stripes in the thicker walled ovarian cysts, and it is clearly due to organisation of blood clot extravasated into the tissue, an occurrence which is undoubtedly very common in ovarian tumours. The accompanying figures, with the explanation appended to them, illustrate some of the more interesting appearances in the parts of the tumour examined (*vide* Pl. V). The description and illustrations of the organisation of thrombi in blood-vessels in Billroth's 'Surgical Pathology' would apply very well to this case, and a long and careful study of my sections makes me incline strongly to the view that while the originally enclosed white blood-cells play an important part in the organising process, their number is greatly increased from some source, and the red blood-cells and fibrine appear to undergo a direct transformation into the areolar network, the result being the formation of a beautiful adenoid form of tissue in place of the extravasated blood exactly similar to that formed in the corpus luteum after the rupture of the Graafian follicle. Time had not allowed any further advance, but it seems fair to suppose that had the patient lived the tissue would have followed the ordinary course of its analogue in the corpus luteum, especially after the birth of the child and the decrease of the vascularity of the parts.

The new-formed tissue seems to vary much according to the number of white blood-corpuscles, the clot originally contained, or contained at the time organisation commenced. Compare parts of figs. 4 and 5 with fig. 6 (*vide* Pl. V). The more white blood-corpuscles the denser and more fibrous is the resulting tissue.

November 2nd, 1875.

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6. *Fibroma of the ovary.*

By W. J. Walsham.

The specimen exhibited is a fibroma of the ovary which was taken from a subject brought to the dissecting rooms at St. Bartholomew's Hospital.

No history of the case was attainable.

The tumour, which is about the size of a small orange, has its origin in the fibrous capsule of the left ovary.

DESCRIPTION OF PLATE V.

Plate V illustrates Mr. Knowsley Thornton's case of Gangrenous Ovarian Cyst. (Page 212) From drawings by himself.

FIGS. 1 and 2 show the extravasation into some of the loose adenoid tissue of the cyst-wall. *a a*. Epithelioid cells. *b*. Pigment masses. *c*. Small blood-vessel full of clot, which is continuous with extravasations through rupture of its wall.

3. Extravasation in firmer fibrous tissue.

4. A clot, with great increase in the number of white blood- or lymph-corpuscles, organizing into delicate lymphadenoid tissue.

5. A small portion of a microscopic cyst full of clot, which is organizing into a similar tissue. *a*. Indefinite granular material, possibly coagulum of original cyst contents, or shrinking of clot.

6. The tissue formed from clot, with a few white corpuscles in it.

Camera × 220 (nearly).

(The dark colour of the white blood-corpuscles and nuclei is due to logwood staining.)

Fig. 1.

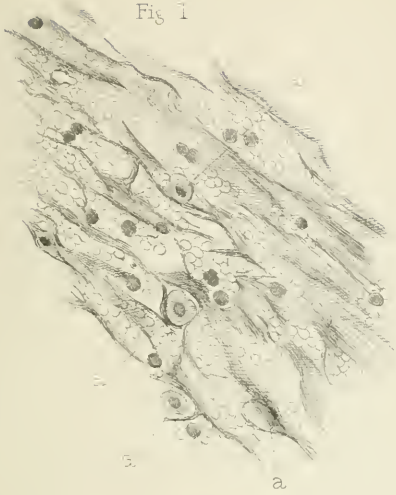


Fig. 2.

Fig. 3.

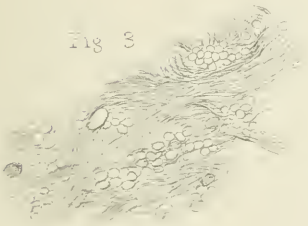


Fig. 4.

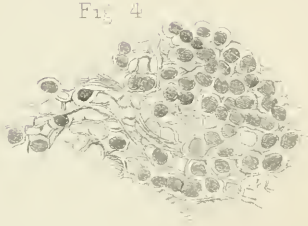


Fig. 5.

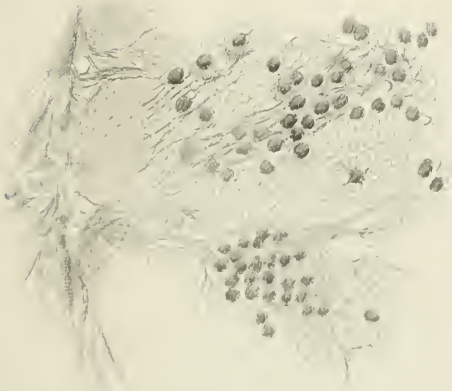
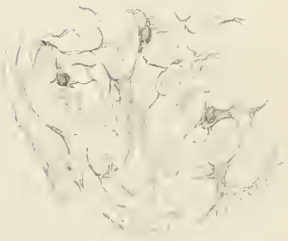


Fig. 6.



Under the microscope it presents the ordinary appearances of the fibromata. The uterus and the right ovary are healthy. The interest about this specimen consists in its undoubted origin from the proper fibrous capsule of the ovary, and not from the tissue of the broad ligament, as is commonly the case with many specimens at first sight supposed to be ovarian in origin. It is a somewhat larger specimen than that exhibited last session by Dr. Goodhart, but, like his, shows unmistakably its origin from the true fibrous capsule of the ovary.

January 4th, 1876.

VI.—DISEASES, ETC., OF THE OSSEOUS SYSTEM.

1. *Ossifying sarcoma of the upper jaw in a boy twelve years old.*

By Howard Marsh.

J. R—, æt. 12, was brought to the Children's Hospital in August, 1875. Five months previously his mother had noticed some swelling of his cheek over his eye tooth. It had gradually, though slowly, increased, but had been quite unattended with pain. On examination a swelling, about the size of a large chestnut, was seen to involve the anterior wall of the right upper maxilla. Its surface was smooth, uniform, nearly hemispherical, covered with healthy mucous membrane, and as firm as bone, or as bone expanded over a firm fibrous growth. At the summit of the mass, however, it seemed doubtful whether some trace of elasticity could not be detected. There was no bulging of the walls of the antrum in any other direction; the right nostril was free from obstruction, and the roof of the mouth and the floor of the orbit were natural. Very firm pressure on the swelling gave him no pain, and he said he had never felt any uneasiness in it. All the teeth were quite sound. The case was watched for a month in the out-patient room, and at the end of this time it was clear that the swelling still increased. He was therefore admitted into St. Bartholomew's Hospital (the wards of the Children's Hospital being under repair). When he was under chloroform the swelling was perforated at its anterior aspect with a narrow gouge, and as it was found that the antrum was the seat of a firm sarcomatous tumour, the superior maxilla was removed entire. This operation (easier in a child than in an adult because the sutures are much less firm) was performed through an incision limited to the middle line of the upper lip and the sulcus between the nose and the cheek, and not prolonged outward below the orbit. Hæmorrhage was only slight. The child recovered so

rapidly that he was up and dressed on the fourth day, and he left the hospital in ten days after the operation.

The tumour was found to occupy the whole cavity of the antrum, and to cause considerable projection of its anterior wall. In structure it was very firm, indistinctly fibrous, and with numerous small masses and thin plates of bone scattered through its substance. It could not be ascertained at what part of the wall of the antrum the growth of the disease had commenced.

Appended is Mr. Butlin's report of its microscopical characters.

November 16*th*, 1875.

Mr. Marsh's case of ossifying sarcoma of the upper jaw.—Microscopical characters.—Trabeculæ of bone traversed all the sections, mapping them out into quadrilateral spaces of various sizes. These trabeculæ contained well-formed lacunæ, in which the bone corpuscles could often be seen, but the canaliculi were not well marked. The trabeculæ were lined most regularly by osteoblasts, as in normal ossification in membrane. The spaces enclosed by the bony trabeculæ contained the true substance of the tumour, consisting of very coarse fibrous material, with numerous cells and nuclei in its interstices, differing from the ordinary tissue of the spindle-celled sarcoma in being much more fibrous. In most of the spaces one or more blood-vessels were seen running through the centre of each space.

Henry T. Butlin.

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2. *Hæmorrhagic periostitis of the shafts of several of the long bones, with separation of the epiphyses.*

By Thomas Smith.

I wish to bring this case, which came under my care at the Children's Hospital, before the Society, for the reason that the disease to be described differs from anything I have before observed, nor do I know of any similar case on record. For the notes of the case from which this account is condensed, I am indebted to Dr. Barlow.

Florence W—, æt. 1 year 11 months, was brought into the Children's Hospital, Feb. 26th, 1875. Was nursed by her mother the first three months and then fed; she has suffered from diarrhœa, but never at any time from any of the ordinary symptoms of syphilis. Neither is there any history of syphilis in her family. The mother is healthy, has had three children at the full time, of whom one died at fifteen weeks from inflammation of the lungs, one is now alive at twelve months of age, and the third is the patient under consideration: the child has been able to stand, but never to walk alone. Eleven months ago both feet began to swell, the swelling gradually spread to her legs and thighs; both lower limbs at that time from the hips downwards seemed to "hang dead and cold," they were not drawn up. She cried when they were touched, there was no external redness; this condition continued for two months, when they gradually began to recover their natural condition; by the summer time the swelling and tenderness had disappeared, and with the exception of some diarrhœa the child was well.

Two months since, the swelling came on again in the feet, and spread upwards to the thighs; a week ago it began to diminish in the feet, while the thighs became rather more swollen. On admission the child was pale and cachectic-looking; slightly rickety about the ribs, has several well-formed teeth, has no symptoms of syphilis; skin sweating, bowels loose, urine said to be very red; has a cough. She lay on her back with her lower limbs stretched out, and quite motionless; there is some œdema of the thighs, and a little about the legs; the appearance of the skin was natural. There was considerable prominence about the region of the trochanters, the thighs being bowed outwards at that point.

On lifting up the limbs there is distinct softish crepitus at the knee and hip joints corresponding with the lines of junction of the epiphyses. The hip- and knee-joints seem to be unaffected: examination does not seem to give the child pain.

Chest.—Has some cough, there is abundance of sharp mucous râles on both backs, the resonance at the bases is impaired.

Abdomen.—Natural, urine could not be saved. Temperature 101·4°. The child was examined by Mr. Smith, who considered that there was separation of the epiphyses, possibly of a syphilitic origin, as there was no sign of suppuration, while the pseudo-paralytic condition and bilateral symmetry were very marked.

DESCRIPTION OF PLATE VI.

Plate VI illustrates Mr. Thomas Smith's case of Hæmorrhagic Periostitis. (Page 219.) From drawings by Mr. Godart.

Fig. 1.

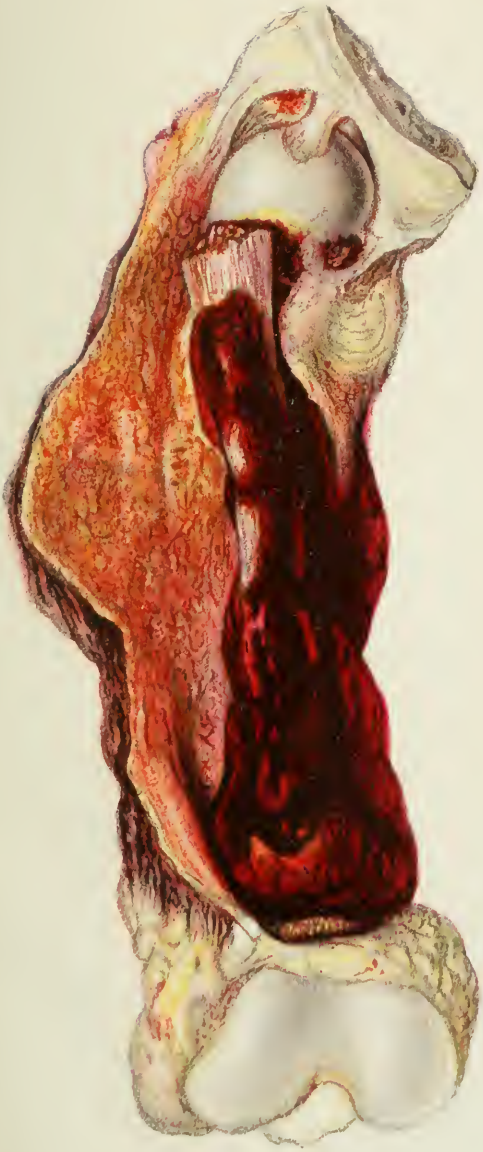
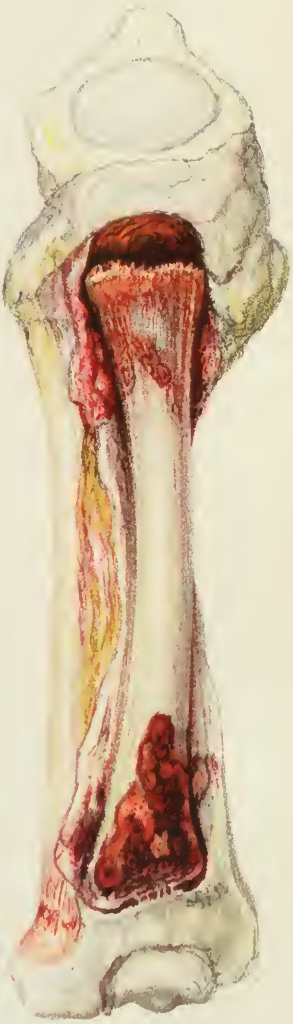


Fig. 2.



Iodide of potassium and steel wine were ordered with suitable diet.

From this time up to March the 4th the child lay much in the same condition, keeping flat on her back and whimpering a great deal; her cough continuing up to the time of her death. At 2 p.m. on March 4th she died suddenly; during her illness the temperature had varied between 99° and 101°.

Post-mortem examination twenty-four hours after death.

Lower limb, left side. Fibres of the gluteus maximus were rather pale, nothing else abnormal noticed in the other muscles in the neighbourhood of the hip-joint. The vastus externus was in a swollen condition, and on cutting into it some blood was found extravasated into its substance, the crureus and vastus externus were in the same condition: the blood seemed pretty uniformly effused through the deeper layers of the muscles; there was no laceration of fibres visible nor any separate clot.

The periosteum of the femur was found separated from the bone in its entire extent; it was intensely injected and thickened. The shaft of the femur was found completely separated from its articular ends at the epiphysial lines and was surrounded by a maroon-coloured clot from a quarter to half an inch in thickness, which was loosely adherent and lay between the periosteum and the bone (*vide* Pl. VI). On removing the clot the surface of the bone, though bare, was quite smooth. No suppuration was to be found anywhere in connection with the bone. Both the upper and lower epiphyses of the femur were quite healthy, the ligaments, cartilages, and synovial membranes of the hip- and knee-joints were quite natural.

Left tibia.—The changes here were of the same nature but less in extent than those in the femur. There was no extravasation of blood among the muscles, but the periosteum about the upper and lower thirds of the shaft was vascular, thickened, and separated from the bone by a thin layer of blood, the shaft was separated from both its epiphyses, but did not lie loose, as the femur; the epiphyses were natural.

The left fibula was affected at its lower end in the same manner, but the morbid changes are less advanced. The ankle-joint was healthy.

In the *right lower limb* there were exactly similar appearances in the femur, tibia, and fibula, and their surroundings as were seen in the opposite limb; the articular ends of the bones and the large joints were quite healthy. The upper limbs were not examined, but cre-

pitus was easily obtained at the shoulder-joint: there was no crepitus at the elbows or wrists, nor at the finger and toe joints. No disease was found in the abdominal viscera, which were all examined, nor was there any thrombosis of the iliac veins.

The heart was healthy.

In the middle of the lower lobe of the left lung was a wedge-shaped patch of congestion, as large as a chestnut, with an abrupt edge, of a reddish-purple colour; coming up to the surface of the lung the pleura over this spot was slightly velvety. There was no softening visible in the mass: in one of the branches of the pulmonary artery in the neighbourhood there was a plug of bright coloured clot; apparently recent. No other patches of the same character were found elsewhere in the lung.

December 21*st*, 1875.

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3. *Tumour of the clavicle.*

By W. J. Walsham.

This specimen, with the following brief notes of the case, was sent to me by my friend Mr. Hancock, of Leicester.

"A. H—, æt. 14, was admitted May 3rd, 1875, an out-patient at the Leicester Infirmary, under the care of Mr. Benfield, complaining of a swelling over the left collar bone, and severe pain in the left shoulder, radiating over the left side of her neck and down the arm of the same side.

"The patient, who was pale but otherwise healthy, stated that she first experienced a dull aching pain in the left shoulder about eight months ago. About a fortnight after this she noticed a slight swelling over the left collar bone, the pain at this time becoming much less and almost entirely ceasing.

"Three months later the pain again became severe, and had gone on increasing, she said, up to the time of her admission, when she was unable to raise the arm without intense suffering. The tumour was also enlarging considerably.

"The family history was good.

"On examination the swelling complained of was found to extend

DESCRIPTION OF PLATE VII.

Plate VII illustrates Mr. Walsham's specimen of Tumour of the Clavicle. (Page 222.) From drawings by himself.

Figs. 1 and 2 show various modes of infiltration of the bone by the sarcomatous growth. 1 in. obj.

Fig. 3 shows infiltration of the bone. $\frac{1}{4}$ in. obj.

4 shows cellular elements of the growth, with some fibrous tissue of the periosteum intact.

Fig. 1.

Fig. 2.

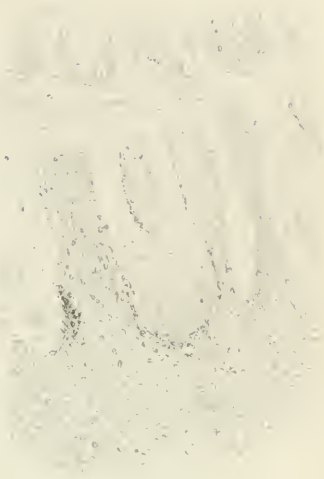

Fig. 3.

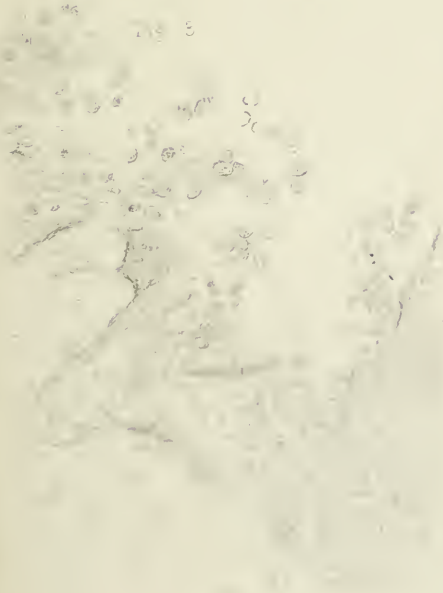

Fig. 4.

from about one inch and a half from the sternal end of the clavicle to the acromio-clavicular articulation.

"Its greatest circumference was at the junction of the outer with the middle third of the clavicle, where it was the size of a small hen's egg. The tumour was continuous with and apparently involved the bone. It was tender on pressure, and firm and resisting to the touch. The skin over it was not adherent, and no glandular enlargement could be discovered. The sternal end of the clavicle was pushed forward and partially dislocated from the sternum.

"June 1st.—The tumour was found to be rapidly enlarging and the glands were becoming affected. Mr. Benfield therefore resolved to remove the growth, and the patient was admitted into the Infirmary.

"9th.—Mr. Benfield excised the growth with the greater portion of the clavicle and several large glands from the neck.

"The operation, which was accomplished with considerable difficulty and attended by profuse arterial hæmorrhage, lasted over an hour and a half. The patient made a good recovery, and when last heard of, three months after the operation, was still in good health."

The tumour, which was sent to me in spirit, was said at the time of the operation to be soft and friable, and difficult to distinguish from the surrounding tissue, it having no distinct capsule or well-defined boundary.

The growth appears to spring from the periosteum, covering the anterior and upper surface of the clavicle. It has no distinct capsule, but seems infiltrating the contiguous bone and muscles. It is oval, with tapering ends, and about the size of a hen's egg. On section it has a whitish colour, is firm and resisting to the touch, and displays a number of fibres radiating into its substance from the periosteum. The outline of the bone can be clearly traced through the tumour.

On microscopical examination the chief mass of the growth is seen to consist of small round cells imbedded in a varying amount of finely granular and indistinctly fibrillated intercellular substance (*vide* Pl. VII). The cells, which appear crowded together without any definite arrangement, have well-defined outlines and contain a considerable amount of finely granular material; they have distinct nuclei in certain portions of the growth, but in other portions no

nuclei can be discovered. Free nuclei are scattered through the intercellular substance in places.

The intercellular substance varies in amount. In places it is scanty, finely granular, and indistinctly fibrillated, whilst in other parts of the growth it is more abundant, and shows well-marked fibrillation.

Bands of fine wavy fibrous tissue running in various directions are seen intersecting the tumour in places, but no true alveolar structure can be made out, what at first sight appears to be alveolation, when seen under a low power, being most probably merely the invasion of the connective tissues by the cellular elements of the tumour.

Sections taken where the tumour is in contact with the bone show the cellular elements of the former irregularly encroaching on the matrix of the latter, the connective tissue bases of the bone, in these situations, being frayed out and distinctly fibrillated. The Haversian canals near the invasion line are greatly dilated and stuffed with the same indifferent material as that forming the chief mass of the tumour, their sides being frayed and fibrillated. Where the canals have been cut obliquely, the continuity of the material filling them with that of the tumour is distinctly seen. Further from the new growth the bone appears healthy.

The tumour has the appearance on the whole of a rapidly growing round-celled sarcoma springing from the periosteum, and involving the soft structures and bone in its immediate vicinity.

The glands removed with the tumour present the same general characters as those of the tumour.

From the age of the patient, the rapid growth of the tumour, the indifferent nature of its structure, and the glandular affection, there can be little doubt of its malignancy.

In the 'Transactions' of the Society only three specimens of tumour of the clavicle are recorded, and these are reported as probably carcinomatous. This specimen appears, therefore, to be the first of its kind as yet exhibited before the Society.

December 21*st*, 1875.

Report by the Committee on Morbid Growths on Mr. Walsham's specimen of tumour of the clavicle.—We have examined the specimen submitted to us and find nothing to add to the description already given by the exhibitor. We regard the tumour as an infiltrating

sarcoma of the round-cell variety, which has originated in the periosteum of the clavicle. Whether it has similar histological characters to those of the three other cases of tumour of the clavicle recorded in the 'Transactions' cannot be determined, owing to the want in those cases of any precise description of the microscopical appearances.

C. Hilton Fagge,
James F. Goodhart.

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4. *Disease of an invertebral substance with double psoas abscess.*

By Jeremiah McCarthy.

A. B—, æt. 40, was admitted into the London Hospital with the following history:—

He had been a railway porter, and had enjoyed excellent health until six months prior to his admission to hospital. He had then, while shunting some trucks, met with some accident, and sprained his back. He had been able, however, to continue at work for five months subsequently, but was then compelled to lie up at home, and after the lapse of a month became so ill that he was brought to the London Hospital.

On admission he had a very large abscess at the upper and outer part of the right thigh, immediately below the great trochanter. This was opened by the house surgeon on duty, and a large quantity of pus escaped. Grating on movement could then be detected in the right hip-joint, but the man's condition prevented any further examination, and he died a few hours after.

At the *post-mortem* examination it was found that the viscera were healthy, but that the right hip-joint was completely disorganised. The capsule was distended with pus, and the cartilages were destroyed, but there was no communication between the joint and the abscess which had been opened by the house surgeon. This proved to be the lower end of a large right psoas abscess, which had burrowed outwards at the level of the lesser trochanter, and so reached the surface at the outer side of the thigh. The left psoas muscle had also been replaced by an abscess which did not extend

below Poupart's ligament. No disease of the vertebræ could be found to account for these abscesses, but a vertical section through the bodies of the lumbar vertebræ disclosed a small cavity partially filled with caseous matter in the centre of the intervertebral substance, connecting the third and fourth lumbar vertebræ. From this cavity two small sinuses passed, the one through the intervertebral substance into the left abscess, the other downward and outward through the body of the fourth lumbar vertebra into the right abscess.

This case suggests a possible explanation of the not infrequent cases where *post-mortem* examination discovers inspissated pus or caseous degeneration in psoas muscles without any evidence of spinal disease or any history of suppuration during life. If A. B— had been suitably treated at the commencement of his illness, his general condition being good, the mischief would probably have ceased, and *post-mortem* examination at a later period might have failed to detect the cavity in the intervertebral substance, or the cavity itself might have been replaced by cicatricial tissue.

April 18*th*, 1876.

VII.—DISEASES, ETC., OF THE ORGANS OF SPECIAL SENSE.

1. *Case of sarcoma of the outer surface of the sclerotic, with commencing invasion of the ciliary body and iris.*

By Edward Nettleship.

This case is of interest not only because it is a rather rare one, but because the microscopical examination gave a clue to the explanation of the patient's symptoms, which could not have been obtained without it; also the directions in which the morbid growth spread could be traced better than usual owing to the early stage at which the eye was removed.

David C—, a healthy looking knife-grinder, æt. 42, came to the South London Ophthalmic Hospital, on August 30th, 1874, on account of his right eye. The ciliary region at the lower and inner part was occupied by a firm tumour of about the size and shape of half a horse-bean. It was seated on the sclerotic, but slightly overlapped the cornea and was covered by conjunctiva; its colour was pale excepting at a single pigmented spot near the most prominent part. The eyeball was congested, T+2; cornea steamy and at one spot infiltrated as if about to ulcerate; anterior chamber, however, of good depth and pupil not dilated; after the use of atropine the pupil dilated to about half its full size, but there were no iritic adhesions. He said that the sight of the eye had begun to fail many months previously, and he believed the eye had been quite blind for two or three months. It had repeatedly been inflamed and painful during the last year, and it was one of these inflammatory attacks which brought him to the hospital. I removed the eye a few days afterwards and found no evidence of external disease excepting the tumour in the ciliary region.

The patient attributed the tumour to two slight injuries of the eye by foreign bodies, received seven years and one year ago

respectively, but was not sure how long it had been growing. That it had been present for at least a year seemed certain, for he went to Moorfields on account of the second injury, and was told there that something was growing and advised to lose the eye at once. As it gave him but little trouble, however, he deferred the operation until the glaucomatous symptoms occurred for which he came under my care. There was no known history of tumours or cancer in his family.

I saw him twenty months after the operation and there was then no sign of recurrence, and he was in good health.

On opening the eye (after hardening in Müller's fluid) I was surprised to find no tumour within it, nor any thickening or other appearance of disease visible to the naked eye in the ciliary body. The growth appeared to be limited to the outside of the globe, and the sclerotic beneath it showed neither thinning nor discoloration, but looked perfectly healthy; it measured 3 mm. (about $\frac{1}{8}$ inch) in thickness. Between the choroid and sclerotic was a large effusion of apparently recent blood, forming a layer which reached from the optic disc forwards as far as the ciliary ligament; at the part corresponding to the tumour, however, the blood did not reach quite so far forwards, showing perhaps that the adhesion between the ciliary body and sclerotic had become firmer at the seat of the primary disease. The layer of blood was thickest at the lower part of the equator, where it reached nearly 5 mm. ($\frac{1}{5}$ inch). The retina was in contact with the choroid, with the exception of a few patches of slight separation.

Microscopical examination showed the tumour to consist of moderately large cells (about $\frac{1}{2000}$ inch wide and $\frac{1}{2000}$ to $\frac{1}{1000}$ inch long) varying in shape from spherical and polygonal to fusiform or nearly spindle shape (*vide* Pl. VIII). They were closely packed and showed only a slight tendency to any regular arrangement. Bands of fibrous tissue from the sclerotic divided the tumour imperfectly into lobules; it was covered by thin but healthy conjunctiva. Although it contained but few blood-vessels the tumour showed no signs of degeneration. The cells generally showed a large nucleus surrounded by a finely granular cell-mass, this being covered again by a thin, more highly refracting layer which was continued into the processes at each end of the cell. Some of the cells near the conjunctival surface were pigmented, but in all other parts they were colourless. In general characters and size the elements of this tumour closely

DESCRIPTION OF PLATE VIII.

Plate VIII illustrates Mr. Nettleship's specimen of Sarcoma of the Sclerotic. (Page 227.) From drawings by himself.

Fig. 1.—A section of the tumour, and of the sclerotic, cornea, and ciliary body, beneath it. The pale shaded parts in the ciliary body, &c., are deposits of tumour-cells. × 12, reduced ½.

c. Cornea.
i. Iris.
s. Sclerotic.
t. Tumour.

2.—A section through the parts in the ciliary region on the side of the eye opposite to the tumour. The suspensory ligament of the iris is crowded with cells. There is a layer of cells on the anterior surface of the iris, and cells also in the sheath of a perforating vessel near Schlemm's canal. × 70, reduced ½.

a. Anterior epithelium of cornea.
c. Cornea.
d. Descemet's membrane.
i. Iris.
l. Ligament. pect. iridis, filled by tumour-cells.
m. Ciliary muscle.
s. Sclerotic.

resembled the cells forming some of the colourless choroidal tumours.

The most interesting features in the specimen relate to the parts secondarily affected and to the probability that the extensions of the growth within the globe, although quite invisible to the naked eye, were the cause of the symptoms for which the patient came under care.

Although the most careful inspection failed to detect any signs of the growth either in the sclerotic, ciliary body or iris, yet with the microscope all these structures were found to be more or less infected; and this not only just beneath the tumour but also on the opposite side of the eyeball, *i. e.*, as far as possible from the tumour. The course of the vessels which perforate the sclerotic beneath the growth is marked out by closely packed lines of cells resembling those of the tumour and situated apparently in the sheaths of the vessels; whilst there are other similar lines of cells in connection with which no blood-vessels can be seen. By this route the cells reached both the ciliary muscle and the venous plexus in Schlemm's canal. From Schlemm's canal they gained access to the reticulated tissue of the *lig. pect. iridis* immediately beneath it. The loose tissue of this ligament seems to have given special facilities for the extension of the growth, for at every point of its circumference it was uniformly and densely infiltrated with the characteristic cells. From this point the morbid growth easily spread backwards to the ciliary body and forwards to the iris (and perhaps to the cornea) (*vide* Pl. VIII, fig. 2, a section taken from the side directly opposite to the tumour). The iris was affected chiefly on its front surface which was almost continuously covered, or closely infiltrated, by a thin stratum of the same cells as far as the pupillary border; this layer was particularly thick at the position of the inner vascular circle, where almost the entire thickness of the iris was more or less infiltrated. Many of the cells, both at the *lig. pect. iridis* and on the front surface of the iris, are seen to be almost loose, one end or the greater part projecting into the aqueous humour (Pl. VIII, fig. 2). This appearance suggests that the iris may have become infected partly by the cells falling off into the aqueous and settling down again at a distant part; such a supposition would, however, seem to be negatived by the absence of any such deposits on the back of the cornea; so that the simple process of travelling along the surface, or in the superficial layers, of the iris seems the most likely

explanation of the origin of the earliest cells in this structure. The growth had also extended along the free surface of some of the ciliary processes and infiltrated the suspensory ligament of the lens, while some scattered cells of the same character occurred on the outer surface of the vitreous about as far back as the ora serrata. In the parts directly beneath the tumour there were considerable islands of the growth in the ciliary process (Pl. VII, fig. 1), and a layer of some thickness extended in the loose tissue between ciliary muscle and sclerotic for some distance further back than the *ora serrata;* the corresponding part of the sclerotic also contained many scattered cells and nests of cells, apparently of the same nature; similar cells were also tolerably abundant in the front part of the choroid, more so, in fact, than in the hinder part of the ciliary body.

The tissue of the corneo-scleral junction and of the cornea for about one third or one fourth of its diameter were, even on the side opposite to the tumour, infiltrated with numerous cells. Many of these closely resembled those of the tumour, only that they were smaller; but I could not with certainty distinguish them from the corpuscles in an inflamed cornea. Close to the edge of the cornea they were usually in rather large groups, and arranged around blood-vessels; further on they were in small groups, or single, or in pairs and triplets. These cells are scattered through the whole thickness of the cornea, but are more abundant in its superficial layers. They come close up to the *anterior elastic lamina*, and at a little distance from the corneal border they actually occur in this lamina itself (fig. 2); they are found at various depths in the lamina, but by far the larger number are close beneath the epithelium, and some appearances suggested their presence even in the epithelial layer, but of this last point I could not be certain. They progressively diminish in size, while at the same time they increase in number as we recede from the edge of the cornea and approach its centre. The largest of them (near the corneal edge) are nearly as large as the cells of the tumour; the smallest (near the centre of the corneal diameter) are scarcely so large as red blood-corpuscles. The presence of numerous corpuscles in the anterior elastic lamina of the cornea is an interesting fact, and is not, so far as I have seen, a usual occurrence in inflammation of this part; it does not, however, seem likely that this peculiarity was specially related, in the present case, to the presence of a sarcomatous tumour near the cornea; it was probably only a modification of the inflammatory process.

The optic disc was rather deeply cupped and its nerve-fibres much atrophied, undoubted signs of a pre-existing glaucomatous state of some standing. There were also some new vessels in the vitreous on the cupped area. The choroid near the disc was œdematous and its veins much congested, but it contained no sarcoma cells. There was thickening, from œdema, of the sheath of the central vessels in the optic nerve.

Although the microscopical examination did not demonstrate the precise mode by which the patient's glaucomatous symptoms were produced, there can be little doubt that they were in some way caused by the invasion of the deep structures at the periphery of the iris and corneo-scleral junction. It is likely that the acute symptoms were due to the intraocular bleeding or to a sudden increase of a previously smaller extravasation. Supposing this to be the case, however, we have still to account for the hæmorrhage, and there is certainly some difficulty in understanding that the obliteration of Schlemm's canal and of some of the anterior ciliary vessels by the invading growth could cause such congestion of the abundant posterior communications with the choroid as to determine their rupture. Still, I do not at present see any better explanation of the facts than this. Many of the deep vessels of the sclerotic at its ciliary part were surrounded by closely packed cells; thus, supposing the eye to have become, as it would be likely to become, everywhere congested by the presence of a rapidly growing tumour, the pressure caused by these perivascular cells in the dense sclerotic might so far prevent the return of venous blood and determine its flow backwards into vessels already overcharged, as to cause their rupture. Another suggestion might be that the growth actually invaded and blocked up some of the large ciliary or choroidal veins, but of this I did not see any evidence.

With regard to the manner in which the previous chronic glaucomatous state had been produced, and its exact relation to the primary tumour and secondary infiltrations, no opinion is offered. Chronic and acute glaucomatous symptoms in the more advanced stages of intraocular tumours are very common, and their occurrence in this case of extraocular growth confirms what from other considerations appears probable, that such symptoms are related, not to the size or character of the intraocular tumour, but perhaps to its position in relation to the choroidal blood-vessels and nerves. It may be noted here that simple inflammatory growths from the sclerotic or episcleral

tissues are not accompanied by symptoms resembling those in the present case, probably because they are not attended with infiltration of the deeper parts in the ciliary region, or, if such infiltration should occur, inflammatory softening of the sclerotic will accompany it, and allow the eyeball to yield to increased pressure from within. *May* 16*th*, 1876.

VIII. TUMOURS.

1. *Colloid carcinoma of the breast.*

By Henry Trentham Butlin.

This tumour was removed from the breast of a woman, æt. 43, who was a patient in St. Bartholomew's Hospital, under the care of Mr. Savory in February, 1875. It was about the size of an egg, had been growing for about twelve months, and presented before removal the ordinary characters of a hard carcinoma. There was not any history of injury, &c., there was no family history of cancer or tumour. The whole of the breast was removed: the patient made a good recovery. Its characters immediately after removal were not sufficiently marked to enable us certainly to decide as to its true nature. It was not encapsuled, but was not in such direct continuity with the mammary gland as carcinoma generally is. On section it presented a coarse alveolar structure of white trabeculæ enclosing spaces containing a juicy yellow or pale orange substance, which was not fluid enough, however, easily to make its escape.

Microscopical examination showed plainly that it was a colloid carcinoma (*vide* Pl. X, figs. 5 and 6). Groups of nucleated cells lay in a transparent substance which separated them from the fibrous trabeculæ forming the boundaries of irregular alveoli. Faint linear markings were occasionally evident between the cells and the trabeculæ. Cells containing a transparent globule could now and then be distinguished. There was not sufficient evidence to enable one to decide whether the colloid change was primary or secondary.

Colloid tumours of the mamma are, I think, sufficiently rare to justify me in placing this case upon the records of the Pathological Society. *October* 19*th*, 1875.

2. *Scirrhus of the male breast. Both breasts affected. Secondary disease of glands.*

By W. W. Wagstaffe.

The man from whom the accompanying specimens were taken was sent to me by my friend Mr. Hague, of Camberwell, in March, 1874. The patient was then 61 years of age, of active and steady habits, and apparently in perfectly good health.

About eighteen months before I saw him he noticed a lump in the left breast, but it did not pain him until six months ago, when it became uncomfortable from occasional pricking and shooting pains: these had considerably increased during the last month so as to prevent his using his left arm in his work. About three months ago he also noticed a small lump in the right breast, but this had not been painful. There was no history of tumour or cancer in the family.

At the time when first seen, March 13th, 1874, the left breast showed a tumour involving the skin to a limited extent below and outside the nipple. Its surface was covered with thinned, red, adherent skin; its substance was of scirrhous hardness, somewhat nodulated towards the nipple, which was slightly retracted. The size of the tumour was about two inches transversely by one inch vertically, and it projected about half an inch from the surface. There was no discharge from the nipple.

In the right breast a small hard mass could be felt, situated just under the nipple, but it was not well defined, and it did not involve the skin.

The axillary glands on the right side were not affected, but on the left there was some suspicion of induration, which, however, was not verified by others who examined him. There was no discharge from either nipple.

On the 25th of March I removed both breasts by semilunar incisions, and the man recovered without any complication, but with two symmetrical scars of the operation.

A year went by and he came to me again (March 30th, 1875), saying he had a lump in his left armpit, which he had noticed about six weeks, and was increasing. He had no pain, no interference

with movement, and had been able to work as a smith since he left hospital.

On examining his left axilla I found a distinct cord running upwards to a hard ovoid mass about the size of a large walnut, and not in the least tender. The scar of operation on this breast was supple and quite free from disease ; and that of the right breast equally healthy. In the right axilla I could feel some small indurated glands high up. His general health was and had been perfectly good.

On April 7th of this year I dissected out all the glands I could discover in the left axilla, and thereby exposed the whole length of the axillary vein up to the clavicle with several of the nerves. One large and several small hard masses were removed and the large mass was evidently scirrhus. A drainage tube was left in and the arm was not removed from the side or the wound uncovered for twenty-one days, at the end of which time it had entirely healed.

The patient was seen last in June, and was in good health. There was no return of disease on the left side. The axillary glands on the right side were rather more distinct and hardened than before.

On making a section of the growth in the left breast, the more superficial portion which involved the skin formed an ovoid mass (about one inch by three quarters of an inch in section) very hard, but bulging on section. This lay over a second mass in which degeneration had evidently advanced further, for the colour and consistence were modified, and placed quite deeply was a distinct cyst apparently due to degenerative changes. Near to the nipple the growth was soft, semi-translucent and almost resembling granulation tissue.

In the right breast a section showed that the nodule was larger than was anticipated. It was larger than a good-sized hazel-nut, very hard, grey, semi-translucent, cupping on section, and the outline was ill defined, owing to the growth running into the fibrous tissues. It lay immediately under the nipple but did not involve it. The removal in each case had been wide of the growth.

The microscopical examination of the tumours (*vide* Pl. IX) has been kindly made by Dr. Creighton, whose account is as follows : "In the section of the small hard nodule of the right breast the most striking appearance is that of the more or less normal acini of the gland. These are generally enlarged and often irregular in form. Their epithelium, which is well shown, remains in situ as in a healthy acinous

gland. In some of the acini—especially the larger of them—there appears to be germination of the epithelium at certain points or along certain lines which gives the surface a ridged and fenestrated appearance. The acini are surrounded by a considerable quantity of connective tissue, which may be described as of three kinds: (*a*) bands of dense tendon-like tissue; (*b*) areas of coarse fibres, closely interlaced and with a few small spaces between them; and lastly (*c*), a tissue composed of finer fibres loosely arranged in an adenoid network, with numerous small round cells in the meshes. The distribution of all these varieties of connective tissue in the tumour in relation to the acini favours the opinion of Waldeyer that the fibrous tissue of mammary tumours is a peri-acinous growth.

"For the larger and softer tumours of the left breast the above description applies exactly, but with the following additions. At certain parts of the growth which presented a caseous appearance to the naked eye, and gave the tumour its soft consistency, the tissue is coloured more deeply by the staining fluid. On closer examination, these deeply coloured areas are made up of masses of cells, ill-defined, with blurred outlines and heaped together irregularly. There is no doubt these areas represent acini, the cells of which have undergone a caseous degeneration."

Dr. Creighton's observations point therefore to the epithelium lining the acini as the starting-points in the development of the new growth, which he recognises as unquestionable scirrhus.

When the large secondary glandular mass removed by the second operation was cut through it gave all the appearance of scirrhus, and to the naked eye exactly resembled the primary growth in the breast.

Microscopically, however (*vide* Plate IX), it presented a somewhat different structure. It may be briefly summarised by stating that an epitheliated growth had invaded the gland tissue; that this epitheliated growth was arranged around and between acini, and gave the appearance rather of dendritic masses lying between open spaces. In these masses the individual cells were not so distinct as in the primary. The margin of the gland was crowded with small cells, but the interior was a mass of epitheliated tissue, of which the cells were peculiarly regular, just as in the primary growth.

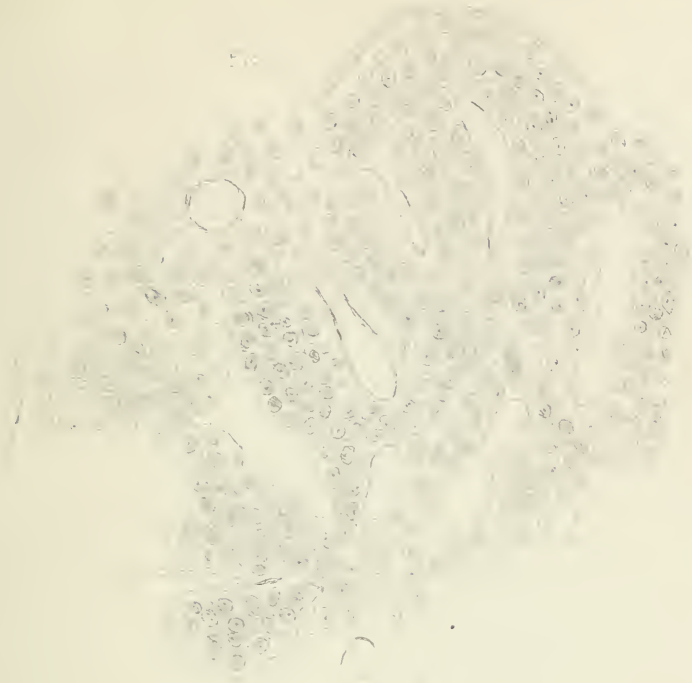
One of the smaller glands removed showed only evidences of inflammatory hyperplasia.

DESCRIPTION OF PLATE IX.

Plate IX illustrates Mr. Wagstaffe's case of Scirrhus of the Male Breast. (Page 234.) From drawings by himself.

Fig. 1.—Secondary disease in an axillary gland, showing an arrangement of indistinctly epitheliated masses between and around open spaces. × 280.

2.—Primary disease in right breast, showing acini, lined with regular epithelium, which appears heaped up in places and thrown into folds. × 280.



The microscopical appearances in this case are given fully by Dr. Creighton ('Report of the Medical Officer of the Privy Council,' new series, No. vi, p. 186 *et seq.*) in his Paper on the "Ætiology of Cancer." It is his opinion that in the more malignant forms of cancerous growths the individual epithelial cells are much less distinctly mapped out than in the slowly growing, and therefore less malignant, forms, and certainly in the present instance this appears to be the case. In the primary growth in each breast the epithelium was remarkably distinct, and in both breasts the disease was of comparatively slow progress. In the right, however, this well-defined tessilation of the epithelium was the most marked, and it was the right tumour which had appeared most slowly, and the glands in the right axilla were either unaffected or very slowly involved. Twelve months after the removal of the tumours, and now, after the lapse of another twelve months, there is only a suspicion of the glands being affected; whereas in the left axilla the glands had become so large twelve months after the removal of the breast growth as to necessitate operation. It is especially worthy of notice that the character of the secondary growth differs chiefly from that of the primary in this respect, that the epithelial cells are much less distinct, the mass of new growth being here composed of epithelium, in which the polygonal outlines are with difficulty made out; the secondary resembles the primary, however, to some extent in its acinous arrangement.

I cannot let this specimen pass into the archives of the Society without referring more particularly to the rarity of cancer in the male breast.[1] I have been able to find only sixty-one cases including those recorded in English and foreign medical literature, and the ten now for the first time published; but casual reference is made to a few more, though in too vague a manner to be of scientific

[1] M. Tauchon in an abstract from the 'Registers of Paris,' extending over eleven years, from 1830 to 1840 inclusive, sets down the number of deaths from cancer at 9118, out of which five were cancers of the male breast. Mr. Birkett in his article on "Diseases of the Male Breast," in 'Holmes's System,' remarks, that carcinoma is the new growth most commonly developed in this organ, that it generally occurs between the ages of 40 and 50, and that it assumes most commonly either the slowly infiltrating or the tuberous form. Other standard authors generally omit all reference to diseases of this structure. Paget, however, in his 'Surgical Pathology' (vol. ii, p. 324), gives 2 per cent. as about the proportion of cases of scirrhus of the breast that are males. No specimens appear to have been submitted to this Society.

value. Cancer of the female breast is, we are all painfully aware, only too common, but even in women cancer in both breasts is extremely rare. Cancer in both breasts in a man has not hitherto been recorded, but the present inquiry has brought to light two other cases besides the one which forms the immediate subject of this paper.

It is to be noticed that cancer of the breast in the male is very commonly infiltrating and disseminated rapidly through the system, but one of the features in the case from which the accompanying specimens were taken, was the excellent health and apparent freedom from further disease in the patient. It seems to me that the occurrence of what appears to be primary cancer at so short an interval in both breasts affords support to the constitutional view of its origin, but why does this not happen oftener, if so ? and the fact of its occurring in a male breast gland bears out Sir James Paget's opinion of its origin in unused or effete structures and organs. But it may fairly be asked, why does not cancer occur more frequently in the male breast ?

In the 40th volume of the 'Transactions of the Medical and Chirurgical Society' is a detailed account of all the cases which Mr. Milton had been able to collect of presumed cancer of the male breast, and if we exclude the eight or ten to which Mr. Liston and M. Velpeau each referred as the number they had probably seen, we can only make a total of forty-two cases. Since the publication of Mr. Milton's paper in 1857 only nine cases have been reported in the journals and numerous works which I have been able to consult, and by the 'Transactions' of this Society no specimen appears to have been exhibited here until the present year, when Mr. Croft and Mr. Maunder each brought forward a case. Nine other cases I have been able to collect from other sources since the exhibition of my specimen, and these I have ventured to include in this summary of cases of cancer of the male breast.

I have drawn up an account of the nine published and ten unpublished cases to which I refer, in order to make this paper a supplement to Mr. Milton's, which is the only one I am aware of available for reference. The details of these cases are therefore appended, and I have added a tabular statement of the total cases hitherto recorded, together with those I have been able to obtain notes of, of which the following is a short summary. Many of the cases are very imperfectly reported, and doubtless in many of them the nature of the tumour

is questionable, but they are recorded as cancer. The total number is sixty-one.

In only thirty-one of these is mention made of which breast was affected, and sixteen of these were right and twelve left, and in three both breasts were affected.

The age of the patient is given in forty cases. The youngest was 25 years of age, the oldest 84. Two were 30 years old, three between 30 and 40, eleven between 40 and 50, nine between 50 and 60, seven between 60 and 70, and six between 70 and 80, so that the largest number of cases occurred between 40 and 50.

The duration of the disease is mentioned in thirty cases, and of these eleven were under one year, eight rather more than one year, one was over two years, three over three years, five over four years, and two as much as eight years.

It is a point of some interest to notice, as indicating the infiltrating character of the growth in the male breast, that at the time when the cases came under observation in more than half the number an open wound had formed. Of forty cases in which a sufficient description is given for us to judge on this point twenty-one had an open wound.

The result of operation is reported to have been successful in twenty out of twenty-three cases that were operated on; whereas of fifteen not operated on twelve are stated to have died shortly. Probably in these last-mentioned cases disease had extended beyond the possibility of interference. Of the successful cases one interesting one occurred under the care of Mr. Cæsar Hawkins, and nine years after operation the patient died without any evidence of return of the disease. Another case was operated on by Mr. Cutler at St. George's three times, but no mention is made of the subsequent progress. The most extraordinary, however, is one recorded by Dr. Warren in his work on tumours, for scirrhous mass is reported to have been removed by him from the right breast of a gentleman 30 years of age, and eleven years after the patient was still well.

Cases of cancer of the male breast recorded since 1857.

November 5, 1859.—In the 'Lancet' of this date are recorded the cases of two patients (male) suffering from cancer of the breast, in the Cancer Hospital, in the year 1856. Both were under Mr. Cooke's care.

Case 1 ('Lancet,' 1859).—A man, A. B—, æt. 45, had a scirrhous nodule in his left breast, noticed for four years. It was movable and at first almond-shaped, but was now small and rounder. It had become reduced in size, not by any particular treatment so much as by non-interference.

Case 2 ('Lancet,' 1859).—A. B—, æt. 67, had the right breast affected for some years with scirrhus, which at first produced enlargement of the gland, and was then followed by the atrophic diminution witnessed in females. The greater part of the gland has now become absorbed, but a distinct lump is felt below and to the right of the nipple. The disease is kept stationary, the general health is good, and no uneasiness is experienced. These two patients are free from suffering.

Case 3 ('Lancet,' 1861).—A. B—, æt. 35, a clerk, was admitted to King's College Hospital in the middle of July, and placed under the care of Mr. Fergusson. About seven months ago a tumour was first noticed in the left breast, and this steadily increased until it involved the whole of the gland, which is now the size of half an orange at its base. There was also a tumour as large as a hen's egg, situated on the anterior margin of the axilla, which was evidently one of the axillary glands, enlarged.

The nature of the disease was so clear that Mr. Fergusson recommended its removal with as little delay as possible, and this was carried out on the 20th of July. The entire gland, including the nipple and fungous growth, was taken away by an elliptical incision, the latter was continued by a single line over the tumour at the margin of the axilla, which also was excised from a surrounding mass of loose areolar tissue and fat. Subsequent examination showed the nature of these tumours to be well-defined scirrhous cancer.

The patient recovered.

There was no past history of blows or other injury having given rise to the tumour; its origin was spontaneous.

Case 4 ('Lancet,' 1861).—A. B—, æt. 52, presented himself in May, 1871, at St. Bartholomew's Hospital, and was placed under the care of Mr. Wormald. He had a small but very bad swelling, superficially ulcerated, in the left breast. From its appearance and by several characters it was diagnosed to be scirrhous. With this eruption the man's general health was good. He stated that twelve

months ago he first noticed a redness of the nipple, which he ascribed to irritation produced by his braces. It had become excoriated about four months before this, but it was entirely free from pain.

Mr. Wormald advised removal of the breast, but the man would not consent to it.

Case 5 ('Lancet,' 1861).—T. L—, æt. 37 (under the care of John Lidderdale, F.R.C.S., Kintbury), robust, and of full habit, had a scirrhous cancer on right nipple. He could not remember having received any injury to the breast nor assign any cause for its origin Mr. Lidderdale immediately removed the cancer by excision. The case progressed well, and in four weeks healed.

Case 6 ('Lancet,' April, 1862).—A gentleman, æt. 72, under Mr. Adams, London Hospital, suffered from what was proved by Mr. Adams and Mr. Middleton to be a scirrhous cancer of left breast. He had suffered for eight months. The cancer was removed on April 8th. The patient's father had died of cancerous tubercles in the liver.

Mr. Adams says that he has collected cases of cancer of *male* breast, and found that the *male breast is obnoxious to every species of cancer to which the female is liable.*

Case 7 ('Lancet,' 1862).—A. B—, æt. 44, was admitted to St. Bartholomew's Hospital, and placed under the care of Mr. Skey, in early part of July, with an ulcerated surface over his right breast, which presented the appearance of malignant disease together with its physical characters. Disease had existed for twelve months. On 12th of July was removed. The patient recovered.

At the same time there were hard glandular swellings in the axilla of the same side, which Mr. Skey did not interfere with, as he was not certain that they were cancerous. The axillary swellings on 26th July were very much diminished.

Case 8 ('Lancet,' 1864).—Charles R—, æt. 60, admitted July 5th, 1864, to the Metropolitan Hospital, under care of Mr. Borlase Childs. A lump about size of walnut in right breast, first noticed about four months ago. July 11th, Mr. Childs removed the tumour and it was found to be cancer. Patient recovered.

Case 9 ('Medical Record,' 1874; Doutrelepont, 'Berliner Medi-

cinischen Wochenschrift,' March 14th).—Scirrhus of male breast. Male, æt. 50; weak; lump noticed 1870 in left nipple, without pain till 1872; ulcerated February, 1873. Mass adherent to ribs in May, 1873; ulceration then $2\frac{3}{4}'$ diameter. Base excavated, cicatrised at one spot and very hard. Several hard knots in skin, and at edge of axilla immovable mass, size of pigeon's eggs. Other nodules over sternum, and axillary glands enlarged and hard. From May to July about a dozen new tumours appeared. Treated by Pot. Chlor. powder.

Two tumours examined microscopically; scirrhus and cell-growths ran along into round lymph tubes between the two tumours.

Mr. George Pollock has kindly furnished me with reference to two cases which have come under his notice.

The first of these was that of a gentleman, Mr. B—, about 65.

Had scirrhus of the right breast, attributed to blow from a stone when addressing his constituents on the hustings. Removed by Mr. Cæsar Hawkins. This patient died nine years after the operation without any return of the disease. This is apparently the same case as Mr. Milton reported, the specimen of which was in the museum of St. George's Hospital.

With reference to this case Mr. Cæsar Hawkins remarks:—

"No surgeon can have seen or operated on many cases of scirrhous cancer in the male breast, as it is uncommon, and of the few cases which have come under my notice I cannot say that I have had an opportunity of knowing the permanent result of operation in any instance so accurately as in the case of this gentleman, who died of apoplexy seven years afterwards without any return of the disease. Cancer in the male breast is, however, obviously different in structure and progress from what takes place in the generality of cases of cancer of the breast in females, and is most like the form in which it shows itself in thin elderly women, of pale complexion, rather than that which is seen in younger women of more sanguine temperament, in whom the tumour grows much larger, and is more rapidly developed than in the former persons. In the male, as I have seen it, the morbid growth is generally confined to the glandular structure and nipple, with fine fibrous covering, and with comparatively little tendency to spread into the adjacent parts, and would, therefore, be expected, *a priori*, to be slow in its progress, and to be more often cured by operation than in most females, which corresponds with general experience as to the disease."

Other cases not previously reported.

Case 10.—The second case is that of Mr. P—, æt. 53, from the West Indies; consulted me for scirrhus of right breast, which he had observed about six or eight months; was removed in August, 1874, by Mr. Pollock, and was well in 1876.

Sir James Paget has also kindly referred me to the following cases which came under his notice and which have not hitherto been published.

Case 11.—The first of these occurred in the practice of Dr. Image, of Bury St. Edmunds, who has favoured me with the following interesting particulars. They have reached me since this paper was presented to the Society, and therefore the similarity of it to my own case could not be referred to in my paper, but it is worthy of notice that in each there was a scirrhous tumour in each breast, appearing in one breast about four months after the other. In Dr. Image's case there was no lymphatic enlargement at the time of the appearance of the breast tumour, and no secondary disease in the axilla as there was in my case, but there was subsequent malignant disease of the skin of one hand.

Mr. Image says: "On October 11th, 1873, I removed a cancerous tumour the size of a walnut from the right breast of the Rev. —, aged 73. As far as I could ascertain, the history of this was as follows:—About fifty years previously he received a blow on his right breast, which became painful, and was followed by a swelling. The swelling became smaller and quite free from pain, but never quite vanished. About three months before I operated on him he was riding a fiery young horse, which reared with him, and he sustained a blow on his right breast. The swelling became painful and began to grow, and in three months' time was as large as a walnut; but the glands in the axilla were not enlarged. There was but little bleeding at the time of the operation under chloroform, but at the end of six hours I was summoned, and found profuse hæmorrhage had taken place. I undid the wound, and found no arterial bleeding, but a profuse general weeping. He had lost a great quantity of blood. He made a good recovery, although in three days after the operation the skin surrounding the wound was swollen and erythematous.

By February 2nd, 1874, a tumour the size of a hazel nut had formed in the *left* breast, which previously had been quite free from

disease; it was of rapid growth, and had attained its present size in about six weeks. I removed it on February 2nd, and left the wound open for several hours. There was hardly any bleeding at the time of the operation under chloroform, but at the end of four hours a general oozing began, which was pretty free, and could only be restrained by hooking up portions of the muscle and tying it; it was venous. Again the swelling and erythema occurred in three days after the operation. Again he made a good recovery.

"There has been no return of the cancer in either of the cicatrices.

"June 1st, 1876.—I operated again on him. This time the cancer affected the skin on the back of the right hand; it came as an aborted boil, but speedily manifested its real nature by rapidly growing. There was no secondary hæmorrhage, but on the third day erythema and swelling. He is now in process of good recovery.

"The family history is interesting. His father, when advanced in life, had a tumour removed from the back of his neck, which formed again in the cicatrix, and was too deep and too extensive for removal, and in consequence of which he died. His nephew died between twenty and thirty years of age of cancer of the tongue."

The next case is one operated on by Sir James Paget in 1871, and again in the early part of 1875 by Mr. Towers, of Brighton, who writes to me that the patient remains well up to the present time.

Case 12.—Mr. Image, senior, of Mildenhall, has also furnished me with the following account of another case. He says in a letter:—

"I well remember another very interesting case. Mr. George Webb, blacksmith, was admitted into the Suffolk General Hospital, February 29th, 1848; no family history of cancer. I knew them all well for two generations. I operated on him for cancer of the (left) breast; he recovered; but after a few months it reappeared and killed him. He would be about seventy now. The exact date of his death is on his gravestone in Whepstead churchyard. His age, therefore, at a guess, was fifty. The disease attacked the pleura and lung subjacent to the breast and so killed him. It had just commenced to ulcerate before I operated. He was a thin spare man, *careful liver*, rather delicate, but otherwise in good health.

The following six cases have occurred at the Cancer Hospital,

Brompton, and I am indebted to the kindness of Dr. Davis, the house surgeon, for the notes of the cases.

Case 13.—Benjamin Parker, ulcerated scirrhus of right breast. August, 1861.

Case 14.—Whitfield Foster, æt. 62. Ulcerated scirrhus of right breast. November, 1867.

Case 15. — Geo. Marshall, æt. 41. Scirrhus of both breasts extending over right side and inside of right arm (ulcerated).

Case 16.—Henry Willmott, married, æt. 55. Extensive ulcerated scirrhus of right breast. No family history. Both breasts large. March, 1872.

Case 17.—Abraham Sly, married, æt. 74. Admitted May 30th, 1874. Suffering from scirrhous tumour, size of a large potato, loose in right breast. Disease began nine months previously. Both breasts large. No family history. Says he received an injury from a fall. Breast removed in June, 1874. Attends as an out-patient. Cicatrix quite healthy.

Case 18.—John Davis, single, æt. 42 years, a soldier. Large ulcerated scirrhous tumour of left breast. Admitted May 17th, 1875, died June 26th, 1875. Says he noticed a small growth, the size of a hazel nut, form near the nipple two years ago which has gradually increased in size, so that the whole breast is now involved. Never received any injury. No family history. Has always been a healthy man previous to present attack. Post-mortem showed extensive secondary deposit in lungs and over pleura.

Case 19.—Mr. Croft's case of sarcoma of the male breast reported in this volume.

Case 20.—Mr. Maunder's case of probable cancer of the male breast reported in this volume.

Case 21.—Case of W. P—, forming the subject of this paper.

November 2nd, 1875.

No.		Age.	Side.	Duration.	Open wound.	Operation.	Result.	Character.	Remarks and *post-mortem* results.
1	Lawrence, Med. Chir. Tr., xvii . .	35	R.	1¼ yrs.	Yes	No	D.	Infiltrating	Lungs infiltrated.
2	Travers „ „ . .	58	L.	4 yrs.	No	No	D.	Infiltrating	Lungs infiltrated.
3	Mus. Coll. S. „ xl (Milton) .	...	...	...	Yes	...	...	...	Approaching true scirrhus.
4	Milton „ „ .	58	L.	1 yr.	Yes	No	D.	Infiltrating	Infiltrating lungs.
	Mus. Bart. Hos.								
5	Paget „ „ .	48	...	½ yr.	No	Yes	R.	Not infiltrating.	
6	Stanley „ „ .	45	R.	1 yr.	Yes	No	D.	...	Bones affected.
	Paget—								
7	Surg. Path. „ „ .	40	...	5 yrs.	Yes	No	D.	...	Skull affected.
8	„ „ „ .	...	...	...	Yes	...	D.		
9	„ „ „ .	52	...	...	Yes	No	D.		
10	„ „ „ .	73							
11	Univ. C. Mus. „ „ .	...	...	...	No				
12	„ „ „ .								
13	Lancet, 1838-9 „ „ .	47	R.	½ yr.					
14	Midsex. Mus.—								
	Lancet, 1841-2, „ „ .	84	L.	1½ yrs.	Yes	Yes	R.	Not infiltrating.	
	Arnott.								
	St. Geo. Mus.—								
15	Brodie „ „ .	...	...	...	...	...	...	Infiltrating.	
16	C. Hawkins „ „ .	64	R.	8 yrs.	...	Yes	R.	...	Died nine years after without return.
17	„ „ „ .								
18	Cutler „ „ .								
19	Johnson „ „ .	30	...	...	...	Yes	R.	...	Three operations performed.
	(Lancet, 1853)								
20	St. Thos., South „ „ .	60	R.	4 yrs.	Yes	...	D.		
	(Lancet)								
21	Simon „ „ .								
22	„ „ „ .	...	...	...	...	...	...	Infiltrating.	
23	Guy's Mus. „ „ .	...	...	...	...	...	...	Not infiltrating.	

24	Guy's Mus.	„	„	...	...	...	No				
25	„	„	„	...	...	...	No				
26	B. Cooper	„	„	41	...	8 yrs.	Yes	Yes	R.		
27	Birkett	„	„	...	...	...	...	...	D.	Infiltrating.	
28	Calloway	„	„	44	...	...	...	Yes	R.		
29	Birkett	„	„								
30	„	„	„								
31	„	„	„								
32	„	„	„								
33	Lancet, 1848, ii, Spry		„	71	...	4 yrs.	...	Yes	R.		
34	„ 1826-7	„	„	25	...	3 yrs.	No	Yes	R.		
35	Farr	„	„	70	L.						
	Liston 8 or 10 cases, and Velpeau 9 or 10	„	„								
36	Syme	„	„	...	...	...	Yes				
37	Velpeau	„	„	...	...	...	...	Yes	D.	Infiltrating	General affection.
38	Blandin	„	„	...	...	...	...	Yes			
39	Degensé	„	„	68							
40	Warren	„	„	30	R.	? 3 yrs.	Yes	Yes	R.	...	Well eleven years after.
41	Weedon Cooke, Lancet, 1859			45	L.	4 yrs.	No	No	R.		
42	„	„	„	67	R.	Some yrs.	No	No	R.		
43	Ferguson	„	1861	35	L.	½ yr.	No	Yes	R.	...	Glands affected.
44	Wormald	„	„	52	L.	1 yr.	Yes	No			
45	Lidderdale	„	„	37	R.	...	No	Yes	R.		
46	Adams, J.	„	1862	72	L.	8 mos.	No	Yes	R.	...	Father died of cancer.
47	Skey	„	„	44	R.	1 yr.	Yes	Yes	R.	...	Glands affected.
48	Childs	„	1864	60	R.	½ yr.	No	Yes	R.		
49	Doutrelepont, Med. Record, 1874			50	L.	3 yrs.	Yes	No	R.	Infiltrating	Treated by Pot. Chlor. powder.
50	Pollock, reported in this paper			53	R.	6-8 mos.	...	Yes	R.		No return two years after.
51	Image, Dr.	„		73	R.	3 mos.	No	Yes	R.		No return two years after.
	„	„		„	L.	3 mos.	No	Yes	R.		„

Cancer of the Male Breast.

No.		Age.	Side.	Duration.	Open Wound.	Operation.	Result.	Character.	Remarks and *post-mortem* results.
52	Image, Mr., reported in this paper .	50	L.	...	Yes	Yes	D.		Cancer of pleura and lung.
53	Cancer Hos., B. P. „ . .	...	R.	...	Yes	No	D.		
54	„ W. F. „ . .	62	R.	...	Yes	No	D.		
55	„ G. M. „ . .	41	both	...	Yes	No	D.		
56	„ H. M. „ . .	55	R.	...	Yes	No	D.		
57	„ J. S. „ . .	74	R.	9 mos.	No	Yes	R.		No return in two years.
58	„ A. D. „ . .	42	L.	2 yrs.	Yes	No	D.		Cancer of pleura and lung.
59	Croft, Path. Tr., 1876	52	R.	3 mos.	No	Yes	D.	Infiltrating	Really a sarcoma.
60	Maunder, Path. Tr., 1876 . . .	44	L.	1 yr.	No	Yes	R.	Skin not affected	No recurrence one year after removal. Nature of growth probably scirrhus.
61	Wagstaffe, Path. Tr., 1876 . . .	61	L.	1½ yr.	No	Yes	R.	Skin affected l.	Subsequent removal of axillary glands.
	„ „ . . .		R.	3 mos.	No	Yes	R.		

3. *Spindle-celled sarcoma in male mammary region.*

By John Croft.

This specimen was exhibited because,

Firstly, it formed an interesting contrast to the case just described by Mr. Wagstaffe.

Secondly, from its peculiar physical signs it appeared to be either a scirrhus cancer or a combination of fibro-plastic growth with scirrhus.

Thirdly, during life it appeared to some surgeons who examined it to be a growth from the mammary gland.

Fourthly, the microscopical examination proved that from one end to the other the growth was a spindle-celled sarcoma, and independent of the gland.

Thomas I—, æt. 52, a labourer, was admitted under my care in St. Thomas's Hospital, on June 24th, 1875. His habits had been temperate. He was a spare man, and in disposition remarkably simple and docile. He observed a small lump above the right nipple about three months ago; probably it had been growing for much longer. It had increased rather rapidly lately, but was not the seat of any pain worth speaking of.

On admission he presented a tumour which measured from above downwards about four inches and five eighths, and from side to side about three inches and a half in the widest part. It ended below at the nipple, and for the sake of description might be divided into three parts. The middle third, which was the first part observed by him, projected boss-like for about one inch and a half, this was firm and elastic in consistence. The skin over it was tense, smooth, and purplish in colour from numerous thin-walled vessels ramifying on it. Around this part the skin was infiltrated, but on each side this condition ended almost abruptly. The upper third also projected, but not so much as the middle. The purple colour was less remarkable. In consistence it was soft and so elastic that many believed it to contain fluid. The integument over it was not infiltrated nor adherent. Both this part and the middle moved pretty freely on the pectoral muscle. The lower third extended to the nipple and was but slightly raised above the level of the skin. This latter was adherent and infiltrated. The nipple was not at all retracted nor was there any apparent alteration of any of the subcu-

taneous tissues at the level of or below the nipple. He did not complain of any pain nor tenderness. The axillary glands could scarcely be felt and were free from any hardness.

After admission the infiltration of integument was observed to increase and a detached hard nodule was found in the skin a little to the sternal side of the central portion. My diagnosis at the time of first seeing him was that the tumour consisted of spindle-celled sarcoma or fibro-plastic material, but just before July 14th I doubted the correctness of this opinion because the infiltration and hardness at the lower part of the growth were very suggestive of scirrhous carcinoma.

On July 14th, with the man's consent, the tumour was freely excised.

When a longitudinal section of the mass was made there was not any creaking noise observed, though the lower part appeared more fibrous than the rest. The middle and upper thirds presented convex surfaces on section, though the lower third exhibited a concave surface on each side. The middle third was rather firm in texture, but the upper one was soft, elastic, juicy, and vascular. There was not any gland structure anywhere visible. After the operation, the wound was dressed immediately with a compress of lint saturated in oily solution of carbolic acid, one to twenty. The man sank on the eighth day after operation from sickness and asthenia.

For a complete microscopical examination and the following report I am indebted to my friend Dr. T. Cranstoun Charles, of St. Thomas's Hospital:

The upper or *soft* portion of the tumour consisted almost entirely of small round and spindle-shaped cells, but with a great preponderance of the spindle-shaped variety. These were arranged chiefly in closely interlacing bundles, and varied greatly in their size as well as in their definiteness of outline, in parts even presenting the appearance of a transparent matrix with regularly arranged elongated nuclei. In addition to a sparing admixture of moderately large spindle-cells there were here and there groups of large round cells and masses of a finely granular nucleated substance lying in the meshes of the small spindle-cell bundles. The skin over this portion of the tumour was much thinned, and apparently replaced by a soft granular material, with numerous irregularly-shaped nuclei scattered through it.

The greater part of the *hard* portion of the tumour was made up of interlacing bundles of spindle-cells, much larger in size than those found in the soft portions of the tumour. Of the cutis covering it the superficial part was quite free from sarcomatous cell-infiltration, but its papillæ were considerably hypertrophied; while in its deeper layers there was a great increase in the number both of the amœboid cells and the connective-tissue-corpuscles, and a considerable enlargement of the lymphatic canalicular spaces. The sebaceous glands were also very large, and in a few points small buds were observed in connection with some of the hair-follicles, apparently as proliferating outgrowths of the outer root-sheath. The deepest layer of the skin was very loose in texture, its interlacing white fibrous bundles appearing hypertrophied, and invading it from below were branching tracts of a soft, finely-granular substance, that varied greatly in appearance at different points. In some places it was in the form of a granular matrix with small round and oval nucleated cells embedded in it; but at others it consisted almost entirely of small round cells. The lymphatic spaces surrounding these branching tracts were in many places filled with elongated clusters of small round cells. In a few points portions of the elongated tracts showed a marked resemblance to lymphoid tissue; oftener, however, the connective tissue was in the form of fine delicate fibres longitudinally interlacing; but, as a rule, there was no fibrillated matrix at all separating the cells.

November 2nd, 1875.

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4. *Discontinuous fatty tumour of the right axillary region.*

By C. F. Maunder.

Joseph T—, æt. 57, admitted into the London Hospital January 26th, 1875.

History.—Noticed a swelling in right mammary region eighteen months ago, which has gradually enlarged to present size—that of a female breast in full lactation. The tumour is soft, elastic, and, as it were, fluctuating, but devoid of percussion wave. The surface is smooth, the skin non-adherent; but on careful manipulation the swelling felt

lobulated towards the axilla. It had been and was painless, but interfered with the free movements of his arm.

The operation consisted in exposing the tumour by an incision into and along its lower border, and then enucleating it. In doing this the mass of fat (weighing 19 ounces) was found to be lying in great measure behind the expanded and attenuated pectoralis major and enclosed in a capsule. The patient recovered.

November 16th, 1875.

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5. *Scirrhus mammæ in a male.*

By C. F. Maunder.

George P—, æt. 44, a publican by trade, of good health though rather a free drinker, experienced about two years ago an itching about the left nipple, which led him to scratch it now and then. Twelve months ago he complained of an aching in the part, soon followed by an excoriation which occasionally yielded a drop or two of discharge of a sanious nature. When seen by me a few days before operation the nipple was surrounded to the extent of the dimensions of a tangerine orange by an infiltration, indurated and inelastic. There was a small scab the size of a shilling upon it. The tumour was not prominent. There was an enlarged, hard, moveable gland in the corresponding axilla.

The mammary tumour was removed September 7th, 1874, in the usual way, and the patient made a good recovery.

November 16th, 1875.—The patient has gained flesh since the operation, weighing now eleven stone six pounds instead of ten stone ten pounds. His general health is good. The scar on the chest is perfectly soft, supple, moveable and healthy in every particular. He can raise the arm well from the side. The gland in the axilla does not appear to be altered either in size or character, and fourteen months have elapsed since the operation. [The patient was exhibited.] *November 16th*, 1875.

Report by the Committee on Morbid Growths on Mr. Maunder's tumour of the breast.—The specimen submitted to us of the breast consists of a breast tumour apparently involving the whole of the gland, surrounded by fat and covered on one side by skin and a retracted nipple.

The specimen has been divided muffin-like so as to show the relation of the tumour to the surrounding fat.

The tumour measures $1\frac{1}{4}$ by $1\frac{1}{8}$ inch, and is situated not immediately beneath the retracted nipple but either above or below it. It is hard, well-defined, and does not spread out into the neighbouring fat along any fibrous cords. It is dark in colour and has apparently been exposed to the air at some time. It extends down to the deepest point of the specimen where the neighbouring muscles have been dried up.

There is no evidence of any proper gland tissue of the breast, and the growth does not seem to have pushed aside any tissues, but rather to have invaded them.

In consequence of the condition of the specimen no satisfactory microscopical observations could be made, but from a careful examination of many slides we are inclined to believe that the growth was an epitheliated one, the epithelium filling irregular spaces. The appearances resembled those usually found in scirrhus, but we are unable to give a conclusive report.

W. Moxon,
W. W. Wagstaffe.

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6. *Epithelioma in chest and toe following the successful removal of an epithelioma of the tongue without involvement of the glands in the neck.*

By Rickman J. Godlee.

The specimen was taken from a man, who first came under notice at University College Hospital in May, 1873, complaining of a tumour on the tongue. He was a strong, well-built man, fifty-six years of age, who had lived rather a hard life, having been a commercial traveller for the greater part of it, and he gave a history such as, I think, is very often obtained in such cases. There was a doubtful history of syphilis. He had been a great smoker, but had always worn his pipe on the opposite side of his mouth. He had had very bad teeth on the side on which the tumour was growing, but they had disappeared long before the tumour showed itself, and there was no family history of malignant disease.

He noticed the tumour six weeks before admission, and it was growing with great rapidity, so rapidly, in fact, that after a few days it was considered advisable to puncture it, in order to ascertain whether or not it contained a cyst. It formed an abruptly projecting tumour, of oval shape, raised three quarters of an inch beyond the surface of the tongue; it did not reach nearly to the tip, and ceased half an inch from the middle line; the finger could easily be passed behind it. The epithelium was complete over the inner part, while the outer was rough, covered with white spots, and probably ulcerated. It was hard and tender, but not painful. No glands were enlarged.

Mr. Erichsen, under whose care the patient came, decided on the removal of that half of the tongue involved by the growth, which was accomplished partly by the galvanic écraseur and partly with scissors. At the end of the operation extensive hæmorrhage occurred from the ranine artery, and a large amount of blood was drawn into the trachea, so that asphyxia was imminent, and was only prevented by the performance of laryngotomy, and sucking the blood out of the chest. Particular attention is directed to this fact because it is thought that in all probability it exerted a very important influence upon the sequel. He made a rapid recovery after the operation, leaving the hospital in about three weeks, but having, as we thought at the time, a little induration in the scar, which, however, ultimately completely disappeared.

He did not reappear until December, 1874, viz. eighteen months after his discharge. His tongue remained perfectly well, and there was no glandular enlargement in the neck; but he now had a tumour on the fourth toe of his right foot, which is one of the specimens handed round. It seemed that he had been quite well for six months after the operation, when he gradually began to feel unwell, without the appearance of any definite symptoms until four months before his readmission, at which time he had an attack of jaundice, and about the same time he noticed some slight discomfort about one of his toes, on which there appeared a small strawberry-like mark which evidently, from his account and his wife's, originated in the skin. It grew rapidly, and was treated with various simple applications, which had no effect upon it. It bled several times. This was four months before his readmission, and it is important to notice that it was not till within five weeks of his readmission that any symptom pointing to anything wrong with his chest was com-

plained of. He then began to suffer from bronchitis. On his readmission, December, 1874, he was much emaciated, sallow, and without any appetite, and suffering from continual slight diarrhœa, and worn out by a very severe cough with copious muco-purulent expectoration.

The tumour on the toe was as large as a small hen's egg, and somewhat of the same shape, with round and everted edges, and a peculiar granular, ulcerated surface, which in itself suggested epithelioma. It was obviously very vascular, and pulsated synchronously with the heart. One gland was enlarged in the groin above it. On the right side of the chest there was a large prominent tumour, projecting around and below the nipple, the tumour, in fact, which is being handed round; it was dull on percussion, firm and elastic, had a weak pulsation, and gave a number of physical signs, with which I will not burden the Society.

There is little to say about the progress of the case; he rapidly sank under the combined effects of the constant pain in chest and toe, the profuse expectoration, the almost complete loss of appetite, and insomnia.

At the *post-mortem* examination it was found that the tumour on the surface of the chest was the prolongation forwards of an enormous mass occupying the substance of the right lung. The lung was adherent to the chest-walls, and the mass involved several ribs and rib-cartilages as well as parts of the pectoral muscles. In the anterior and posterior mediastina were large masses of the growth springing from the lymphatic glands surrounding the large vessels, and pushing the heart over to the left, but which was separated from the large tumour by a mass of condensed lung slightly if at all affected by the growth. The heart itself was completely adherent to the pericardium, and in it were a number of nodules of the growth which apparently involved the pericardium only, and did not extend into the muscular substance. The only other secondary deposits were two or three nodules in the left lung, and in the lymphatic gland in the right groin previously mentioned. The tongue showed no trace of return, and a prolonged search in the neck failed to detect any enlarged lymphatic glands.

I drew attention to the thickened lung tissue remaining between the mass in the lung and that in the mediastina, because it seems to show that they are distinct developments, viz. that the former did not originate as a direct prolongation of the latter. There are, how-

ever, a few small masses apparently round the bronchi in this condensed tissue.

The chief interest of the case is, I think, the problem as to the method in which the system became affected from the original growth, if it did so at all. If we agree that the growth is epithelioma, and I think it is impossible not to do so if the specimens I have put under the microscope be examined, it must be allowed that the primary tumour was that removed from the tongue, for a primary epithelioma of the lung would be so rare as to be almost impossible.

If, then, it occurred primarily in the tongue we must decide whether the recurrence occurred before or after the operation for the removal of the primary growth. Any one who would maintain that the recurrence occurred subsequently would of course be an advocate, and a strong one too, of the constitutional character of the disease; but it seems to me that he would prove too much, for he must suppose the patient to have a tendency not only to the development of cancer, but of a particular kind of cancer, viz. epithelioma, which most of us would probably hardly like to admit.

But supposing it occurred before or at the time of the operation I can see only two natural explanations of its rationale:

1st. That the system was affected without any glands being involved.

2nd. That pieces of the tumour were drawn into the lungs, and so formed the nucleus of a new growth.

The first of these, though of course it is recognised as a possible one, is opposed to all accepted notions as to the usual method of the propagation of epithelioma; and the second may appear at first rather a wild theory to hold when there is no more proof than in the present case. It is, however, I think, the most likely explanation, because in the first place the other is so unlikely, and in the second the accident which occurred during the operation would afford an excellent opportunity for pieces of the tumour to be drawn into the lungs.

It may be said, "Why if this is a possibility at all do patients with epithelioma of the tongue, as a rule, escape so obvious a danger?" To this I would reply—"In the first place accidents such as the present are not of common occurrence during the removal of tumours of the tongue, and in the ordinary course of events the opportunity for *living* masses of the tumour to pass into the trachea is not very great. For the pieces which are constantly being separated

from the ulcerated surface of an epithelioma are separated by a process of sloughing; in other words, are dead and therefore incompetent to form the starting-point of a new growth, even if they reach the lungs; and lastly, even supposing living particles were separated from the growth they would be much more likely to pass into the œsophagus than into the trachea, when it is evident that their chance of development would be *nil*.

The analogy of this method of propagation is, I think, not far to seek. It is a close imitation of what occurs when disseminated cancer springs up in the peritoneum as the result of rupture of a larger mass.

Considerable support is given to the idea by a case reported in our 'Transactions,' "Epithelioma of the Œsophagus Ulcerating into the Trachea," by Dr. Moxon, in 1869, vol. xx, p. 28, in which, however, the proof was much stronger than it can be in my case.

December 21*st*, 1875.

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7. *Sequel to Dr. J. Swift Walker's case of recurrent fibroid tumour of the anterior portion of the lower extremity, recorded in vol.* xxii, *page* 243, *of 'Pathological Transactions,' and in vol.* xxiv, *page* 209 *et seq.*

By W. Spencer Watson.

December 31st, 1872.—In consequence of the recurrence of the tumour situated between the tibia and fibula which is now the size of an orange and continually bleeding, it was decided to amputate the leg at the lower third of the thigh.

The wound healed up well and the patient seemed entirely to recover, getting about to do her household duties on a pair of crutches.

September, 1873.—Miss ——— again visited me; she had a little enlarged gland, as she supposed, in the groin, just external to the scar where the femoral artery was tied. It is about the size of a walnut, very movable, tender to the touch, and giving her a little pain if she jerks the stump. Does not interfere with sleep. Health very much deteriorated since this growth appeared, as it preys upon her mind that the tumour is returning. She wanted it removed,

but I was afraid of doing so, thinking very probably all the inguinal glands were affected.

She went to Manchester by the advice of her relations to see a surgeon there, who agreed with me "to let it alone," at all events for a while.

November 26th.—She fell down stairs and hurt the lump in the groin. It is now as large as an orange, with many tortuous blue veins crossing over it and the skin very tense and hard. She states that it is growing so rapidly that day by day she can perceive the difference in its size.

November 29th.—The whole tumour has come away in one large slough, leaving a large open sore filled with shreds of sloughing tissue hanging loose. During the night there have been several attacks of hæmorrhage. The size of the open sore measures four inches by three, being about one inch deep. Patient blanched and evidently sinking.

December 10th.—An attack of hæmorrhage came on and she sank. In the course of the three and a half years during which the case lasted five operations were performed for the removal of tumours at the site of their primary growth. The femoral artery was tied and amputation performed. *December 21st*, 1875.

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8. *Recurrent epithelioma of chin and submental tissues removed by operation.*

By Christopher Heath.

The patent, æt. 55, had an epithelioma of the lower lip successfully removed in 1872. In November, 1874, he noticed a lump on the chin, which rapidly increased in size. In November, 1875, when admitted into University College Hospital, the growth was of the size of a small cocoa-nut, measuring 6 by $5\frac{1}{2}$ inches, and extending $2\frac{1}{2}$ inches to the right and $3\frac{1}{2}$ inches to the left of the middle line and reaching down to the hyoid bone. The chin was adherent, and at the lower part were six ulcerated openings, through which a fetid discharge constantly exuded. Mr. Heath removed the growth by sawing off the chin without opening the cavity of the mouth and then dividing the soft tissues down to the hyoid bone with the galvanic écraseur. The man made a good recovery and was ex-

hibited to the Society with the wound nearly healed. The tumour is firmly adherent to the section of the body of the jaw, the line of which is unbroken and the surface healthy. On cutting into the under surface of the tumour a cavity as large as an apple is opened, full of fetid ichorous fluid, with irregular walls in which are six sinuses.

Examined microscopically by Mr. Gould, the growth is seen to be a typical example of globular epithelioma with very numerous globes quite filling the field, and this tissue extends quite to the lower border of the growth near the hyoid bone. In front there is no epitheliomatous tissue within half an inch of the line of removal, but the muscular tissue of the chin is here infiltrated with a small-celled inflammatory growth. *December* 21*st*, 1875.

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9. *Cancer of both breasts and ovaries.*

By Sidney Coupland, M.D.

For the following notes of this case, which is of great interest both clinically and pathologically, I am indebted to Mr. Hulke, who has kindly permitted me to bring these specimens before the Society.

"E. W—, æt. 24, a rather stout, good-looking Irish brunette, was admitted into Regent Ward of the Middlesex Hospital in April, 1875, having then in her right breast, mostly lying in its sternal half, a very hard subglobular mass, about three inches in diameter, with an ill-defined border and slight adhesion of the skin at its most prominent part. Between this mass and the sternum were two scars of an abscess which, seven years ago, followed her first and only confinement. In the upper part of the breast was a small hard knot, separated by a short space from the chief mass and only obscurely connected with it by an intermediate hard line. In the armpit there was a small, movable, and not hardened lymphatic gland. The nipple was flat; she said it had always been so, and that owing to this she had not nursed with this breast.

"She menstruated regularly, and her appearance was that of a person in vigorous health. None of her relations had to her knowledge had tumours of any sort.

"April 22nd.—The whole breast, including the outlying knot and also the axillary gland, were excised with antiseptic precautions, and the wound was dressed antiseptically.

"30th.—A slight attack of erysipelas began, from which she was convalescent in about a week.

"May 28th.—A small, hard, red, tender knot has appeared over the third intercostal space. It had the aspect of a little abscess threatening, and it disappeared entirely a few days later.

"On the 11th June she went home convalescent.

"Towards the close of September, during Mr. Hulke's absence, she was readmitted with two small recurrent knots in the right mammary region, which were removed by Mr. Clark.

"In November a sudden and rapid evolution of secondary knots took place, beginning in the right mammary region; next the left breast became, as it were, infiltrated with a diffuse scirrhous growth, which quickly made it a large, hard, stiff, hemispherical mass. Simultaneously with this there was a rapid dissemination of scirrhous knots through all the soft tissues over the front and sides of the chest, converting them into an inflexible brawny cuirass, and while this went on the lymphatic glands in both armpits and above the collar-bones became infected. In less then a fortnight from the beginning of this outburst her chest had become quite fixed. She was now unable to lie down; her dyspnœa was terrible to witness. She was quite cyanosed and died suffocated."

Before detailing the appearances found at the *post-mortem* examination I would venture to advert to the most striking features presented by this clinical history. There is, firstly, the occurrence of cancer in the breast, which had never been used for lactation, and had in consequence been previously the seat of inflammatory changes. There is the early age of the patient (see below) and her robust and healthy appearance, retained even to the end of her fatal malady, and the absence of any hereditary taint. More remarkable still is the quiescence of the disease after the operation in April, an operation which for nearly six months could be regarded as being perfectly successful. In September, however, a few recurrent nodules appeared at the primary seat, and were removed. Then two months later occurred the sudden outburst of cancer, by which the fellow-breast was involved, and the whole soft tissues of the thoracic wall rendered so firm and rigid as actually to lead to a painful death from suffocation. The rapidity with which this condition was evolved

is not often seen, but doubtless it was in great measure due to the well-nourished and full-blooded condition of the patient. Mr. Hulke has informed me that in all his experience he has never seen so rapid a recurrence and extensive diffusion of cancer take place within so short a time.

Turning now to the *post-mortem* examination, the extremely well-nourished condition of the body was noticed. A thick layer of healthy-looking fat occurred in the abdominal parietes, and the omentum was laden with fat.

The skin covering the upper half of the thorax was thickly beset by firm adherent nodules, extending over the clavicle and the right side.

The site of the right mamma was occupied by a deep puckered triradiate cicatrix, while the left breast was converted into a firm, hard, hemispherical mass, the nipple not being retracted, and the surface nowhere ulcerated. The thick layer of subcutaneous fat of the thoracic parietes was unusually firm and crisp; it cut with a peculiar "crunching" sound, and it was rendered so rigid and inflexible that, in order to the reflection of the soft parts from the ribs, it was necessary to make transverse incisions through the wall, both above and below. The soft tissues, both fat and muscle, were then seen to be the seat of diffuse cancerous infiltration, chiefly in the form of firm white bands or cords, which on section appeared as nodules of the size of peas. A vertical section was made through the left breast and the subjacent tissues, and two of the slices thus obtained are before the Society. It will be seen that the form of the gland is completely retained, and to some extent its structure, only it is much indurated, and presents a uniform opaque white colour from cancerous infiltration. Completely surrounding the enlarged gland is seen a layer of fat studded by firm opaque white nodules, and this again on the surface is covered by the skin, of which the true layer is much thickened and milky-white in appearance. The specimen shows also a portion of the greater pectoral muscle studded with cancer nodules.

The microscopical characters of the infiltrated parts were those of scirrhous cancer, everywhere surrounded by an abundant infiltration of small round cells, which were especially well seen in the connection between the fat-cells and muscular fasciculi.

There was no implication of the ribs or the parietal pleura in the cancerous infiltration. On the left side the pleural cavity contained

a large quantity (not measured) of dark straw-coloured fluid,[1] and several shreds of lymph passed between the pleural layers. The lung was condensed and airless. On the right side the pleura covering the apex of the lung was converted into a firm white layer, but there were no pulmonary nodules, and the costal pleura was unaffected. The posterior mediastinal glands were enlarged, white, and cancerous.

The visceral layer of the pericardium was also apparently infiltrated, a number of slightly raised white patches, irregular in shape, size, and distribution, occurring on both surfaces of the heart, contrasting with the rich yellow fat with which the organ was well covered. Just as in the pleura, the parietal layer of the pericardium was free from deposit and perfectly smooth.

There was no peritoneal cancer, *nor were any of the lymphatic glands within the abdominal cavity affected;* none were even enlarged. The liver, spleen, and kidneys were natural.

In the pelvis, however, it was found that the cancer had attacked the ovaries symmetrically, and so symmetrically that in size and appearance these organs differed hardly at all from each other. Each was enlarged to the size of a chestnut, was adherent to its Fallopian tube, and presented slight lobulation. On section the ovary was soft, of a pure white colour throughout, presenting no traces of normal structure. Microscopically it presented the characters of a medullary cancer, the stroma being reduced to a minimum. The uterus was of small size.

Beyond the rapid diffusion of the cancer locally, the question which seems to me of greatest interest—which, indeed is the main object of my showing the specimen—is the association of cancer of both mammæ and of both ovaries, without any direct continuity of the disease between the two sites. To evoke the fact of the physiological sympathy of two such widely removed organs to explain such a case as this is a view perhaps too fanciful to be entertained; but yet it is difficult to put such a consideration entirely out of sight, since the date at which the ovaries were attacked must have been, from their characters, coincident or nearly so with the recurrence of the disease in the mammæ. On the other hand, ovarian cancer secondary to mammary is by no means frequent, if we

[1] It has been suggested, and with much truth, that the amount of pleural effusion no doubt aided greatly in the mode of death, over and above the fixation of the chest-wall by the cancer "en cuirasse."

may judge from the experience afforded by the cancer wards of the Middlesex Hospital. I append below a table compiled from the annual reports of the Surgical Registrars[1] of this hospital from the year 1867 to 1874 inclusive, together with the results of the present year's experience, limiting myself to those cases which were examined after death. The table includes eighty-nine cases of mammary cancer in the female, and shows the relative frequency of secondary implication of the viscera. It will be seen that, whereas the liver was affected in thirty-five cases and one or both lungs in fourteen, the ovaries were attacked but five times, in three bilaterally and in two one organ (the left) only. In one case, that of cancer of the left mamma, the only other regions noted as being attacked besides the ovaries were the axillary glands and the pleuræ; in one the bones were also the seat of cancer, and in the remaining three cases there was more or less extensive affection of the abdominal viscera in addition to the ovarian cancer, viz. in one case the liver, in a second the spleen, suprarenal capsules and retroperitoneal glands, and in the third case, in which both breasts were attacked and one ovary, the liver and mesenteric glands.

The age of this patient is worthy of notice. She had borne a child it was true, but she was only 24 years old, and I find from the records of the Middlesex Hospital that out of 235 patients suffering from "mammary cancer" only three were below 30 years of age, the youngest being 26 years. Of the remainder there were between the ages of 31 and 40 years forty cases, between 41 and 50 eighty-eight cases, 51 and 60 seventy-two cases, 61 and 70 twenty-six cases, and over 70 nine cases. *January 4th*, 1876.

P.S.—Mr. Waren Tay has kindly drawn my attention to a case of "acute symmetrical carcinoma" under the care of Dr. Fraser, recorded in the 'London Hospital Reports,' vol. iii, p. 303. The patient was a servant girl, twenty years of age, who was admitted into the hospital in October, 1865. The whole illness dated but two months, and not only were the mammæ and ovaries symmetrically affected, but the lymphatic glands all over the body, as well as the heart, liver, kidneys and sternum, were the seat of secondary deposit.

In the very wide diffusion of the new growth this case (and others) differs from mine, of which the remarkable feature is, that, beyond the ovaries, no pelvic or abdominal organ was attacked with the disease.

[1] H. Arnott, H. Morris, and A. Clark.

Analysis of eighty-nine cases of mammary cancer examined after death, showing relative frequency of the seats of secondary growths. Compiled from the 'Reports of the Surgical Registrars to the Middlesex Hospital,' 1867 to 1875.

Mamma affected at time of *post-mortem* examination.	Right . . 31 cases.	Left . . 41 cases.	Both . . 17 cases.	Total 89.	Per-centage.
Secondary growths in—					
Axillary glands[1] . . .	in 28 cases . .	in 41 cases . .	in 12 cases . .	81	91·
Other lymphatic glands . .	11 „ . .	13 „ . .	1 case . .	25	28·1
Pleura	7 „ { R. side 3 / Both 4 }	8 „ { L. side 4 / Both 4 }	6 cases. Both sides	21	23·6
Pericardium	1 case . .	0 „ . .	1 case . .	2	2·2
Peritoneum	1 „ . .	1 case . .	1 „ . .	3	3·4
Lung	6 cases { R. side 2 / Both 4 }	8 cases { L. side 4 / Both 4 }	0 „ . .	14	15·6
Stomach	0 „ . .	0 „ . .	1 „ . .	1	1·1
Liver	11 „ . .	20 „ . .	4 „ . .	35	39·3
Pancreas	0 „ . .	1 case . .	0 „ . .	1	1·1
Spleen	0 „ . .	2 cases . .	0 „ . .	2	2·2
Kidney	0 „ . .	2 „ R. side .	0 „ . .	2	2·2
Supra-renal capsule . . .	0 „ . .	1 case. Both sides	0 „ . .	1	1·1
Ovary	1 case. Both sides	3 cases { L. side 1 / Both 2 }	1 case. Left side	5	5·5
Uterus	1 „ . .	0 „ . .	0 „ . .	1	1·1
Bones	5 cases . .	0 „ . .	0 „ . .	5	5·5

[1] Only those structures are included here which are mentioned as being affected in the lists from which this table was compiled.

10. *Tumour of the femur.*

By Jonathan Hutchinson.

The subject of this case was a fairly healthy looking man of about twenty-six. There was no history of malignant disease in his family. For about fifteen months he had been aware that there was something wrong in his right thigh. At first there had been a good deal of pain in the lower part of the bone, this was soon followed by evident thickening, and for the last eight months the existence of a tumour had been obvious. He had been seen by several distinguished surgeons in private, and in the suspicion that it might be of syphilitic origin iodide of potassium and mercury had been tried. When he came under my care the tumour was too large to permit of much doubt. It was very firm, as large as two or three fists, and embraced the bone in its lower third, adhering firmly to it. Amputation was clearly the only resource.

After removal of the limb the tumour was found to spring from the bone and to involve its medulla. The bulk of it consisted of large, smooth masses quite external to the bone, which were encapsuled by cellular tissue, and which displaced the muscles. There was no tendency to infiltration of the soft structures, which were simply pushed aside. The bone was not expanded, but its surface over a considerable extent was eroded deeply, and its medullary cavity was occupied by growth exactly like that which occurred externally. The two were, in fact, continuous through the bone at various parts. On section the tumour was almost homogeneous, of an opaque white appearance like hard blanc-mange.

The following are the particulars of the examination of the tumour as prepared for me by my friend Mr. Nettleship.

Scrapings from the fresh tumour were composed chiefly of cells, but there was a variable quantity of connective tissue in bands and fibres intersecting the growth. The great majority of the cells were round, or nearly so, only a few being fusiform or spindle-shaped. They varied much in size, the largest being from two to three times as wide as a red blood-corpuscle, but the majority were not equal in diameter to more than $1\frac{1}{2}$ corpuscle. Many were nucleated, and some showed a definite nucleolus. Many of the cells were undergoing fatty degeneration.

Sections made after hardening were composed of a wide and irregular mesh work (alveoli) of fibrous tissue in fine fibres and broader bands, stuffed with cells. The relative quantity of the two elements varied much in different parts. In some parts there was little but ordinary white fibrous tissue, between the strands of which single or double rows or oval collections of cells were more or less thickly placed. This condition, apparently the earliest, passed rather suddenly into one in which the greater part of the mass was cellular, the fibrous alveoli being comparatively insignificant, while in some parts it was not easy, except in brushed specimens, to see any fibrous framework at all. The alveoli were, I think, always fibrous, and not composed of separable spindle-cells; they often, however, showed large oval nuclei, and gave the impression of having been distinct cells which had become fused together.

The cells of the tumour in stained sections were easily divisible into two groups:

a. Well-formed rounded, oval, or polygonal cells, varying in diameter from $1\frac{1}{2}$ to 3 blood-corpuscles, often nucleated, and staining well with logwood; these formed, as already stated, the bulk of scrapings from the fresh tumour.

b. Large numbers of smaller cells, less regular and definite in outline, granular, more or less shrivelled, and not staining at all with logwood. These were, no doubt, degenerating examples of the larger cells; they were most abundant in the parts of the tumour which contained least fibrous tissue, *i. e.* presumably in the oldest parts, and were almost entirely wanting where the fibrous septa were abundant.

The tumour appears to consist of a growth of cells in the fibrous tissues close to the bone (probably the periosteum); the alveolar portion of the growth probably represents this fibrous tissue, much expanded and subdivided by large groups of cells, and is not a new growth of fibrous matrix. The alveoli are of large size, and contain large clumps or masses of cells, excepting at those parts where the growth is beginning; there is not anywhere a fine reticulum separating the individual cells from each other, or dividing them up into small groups. The tumour contained a moderate number of capillary vessels.

The man died about ten days after the amputation, having had symptoms suggestive of pyæmia, but by no means positive.

The following are some brief particulars as to the autopsy:—

The femur cut across above the part from which the tumour grew showed no positive changes; the periosteum was stripped off for about one third of an inch at the cut end, the bone was here discoloured, but probably only *post-mortem*. The femoral vein from the stump, traced up a little further than the brim of the pelvis, was healthy. The femoral artery in the stump contained a clot, which was soft at the distal part, but healthy and adherent to the coats of the vessel at the middle and proximal parts. In the upper parts of the artery were several thin shreddy bits of yellowish fibrinous substance slightly adherent to the vessel; they were found chiefly at the orifices of branches, and consisted of white corpuscles and fibrine.

There were deposits (thromboses) in the spleen. Kidneys healthy. The right lung was pneumonic, the left congested.

Microscopic examination of parts obtained at the autopsy (Mr. Nettleship).—The lymphatic glands examined were—

1. A rather firm femoral gland, which in section, when fresh, looked healthy. Scrapings from it showed, besides ordinary lymph-cells, numerous much larger cells with large nuclei. Sections made after hardening were composed to a great extent of cells considerably larger than white blood-cells, varying indeed from one and a half to twice their diameter, closely packed and often very definitely angular from pressure. Usually when seen in very thin sections or tags these parts of the gland showed no reticulum whatever. In some other parts a reticulum was made out more or less distinctly. I think these cells were morbid. Other reasons for thinking the gland diseased were—(1) Many of the arterial branches at the hilus of the gland contained within their cavities a stratum of nucleated cells several layers in thickness and attached to the *intima*. (2) The adipose tissue attached to the gland was much infiltrated with cells which formed collections of various sizes between the adipose cells, and in some parts completely surrounded them. (3) There was much blood extravasated into the gland in lines and occasionally in patches, and some of this had become converted into rusty granules.

2. A gland from the pelvis was examined by fresh scrapings. It was very soft and contained more of the large cells and fewer lymph-corpuscles than the femoral gland. There were also numerous spindle-shaped cells, so uniform in character as to raise a doubt as

to their being merely the branching cells of the reticulum broken down by scraping.

The nodule in the liver, immediately beneath the peritoneum (supposed to be pyæmic), was well circumscribed by compressed liver tissue. It consisted of immensely distended blood-vessels (capillaries probably) separated by greatly compressed liver-cells. A large vessel entered it from its peritoneal surface. It contained no cells like those in the tumour and was probably of embolic (pyæmic) origin.

The deposits in the liver were all immediately beneath the peritoneum and reached deeply into its substance; they were abruptly circumscribed, almost encapsuled, having a uniformly rounded outline (excepting the peritoneal portion) and their surface became convex on section. Their texture was granular and soft, but not softer in the centre; the colour uniformly red and with no trace of stratification or division into layers of different colours. Scrapings of the fresh deposit gave nothing but blood- and liver-cells and free nuclei (probably of liver-cells), but there was fatty degeneration in one portion. *January 4th*, 1876.

Report by the Committee on Morbid Growths on Mr. Hutchinson's tumour of the femur.—The specimen submitted to us consists of the lower end of the femur surrounded by a soft white tumour. The bone beneath the tumour is considerably increased in size. The compact tissue is increased in thickness and less dense than natural. The medullary canal is filled for a distance of about $2\frac{1}{2}$ inches with very hard and dense cancellous bone. Scattered through this bone are two or three nodules of soft white substance, exactly resembling the subperiosteal tumour. The subperiosteal portion of the tumour can be easily peeled off the bone like the periosteum in periostitis, and beneath it are found irregular nodules and spicula of bone resembling those ordinarily seen in chronic inflammation. The periosteum cannot be recognised, being absolutely lost in the tumour. The thickening of the bone is not strictly limited to the region of the tumour, but extends beyond it for some distance. The tumour is soft and now (at least forty-eight hours after removal) yields an abundant milky juice on scraping. On examining sections from specimens hardened in alcohol it is found in its fully developed parts to be composed of densely packed masses of round and oval nuclei, each having one or more bright shining nucleoli (*vide* Pl. X, figs. 1, 2, 3 and 4).

DESCRIPTION OF PLATE X.

Figs. 1, 2, 3, and 4 illustrate the Report by the Committee on Morbid Growths on Mr. Hutchinson's specimen of Tumour of the Femur. (Page 268.) From drawings by Mr. Marcus Beck.

FIG. 1. Shows the peculiar alveolar stroma obtained from some parts of the tumour by washing away the cells. The stroma is almost structureless. × 250.

2. Another part of a washed specimen, showing that the cells are not in alveolar spaces, but embedded in the stroma. × 250.

3. Cells and stroma. Stroma almost homogeneous. There are a few fibres. × 500.

4. Loose cells, showing irregular masses of protoplasm surrounding the nuclei. × 500.

Figs. 5 and 6 illustrate Mr. Butlin's specimen of Colloid Tumour of the Breast. (Page 233.) From drawings by himself.

FIG. 5. Groups of cells, one of which contains colloid material, lying in colloid. × 260.

6. A section of the tumour seen with a low power.

Fig. 7 illustrates Dr. Peacock's case of Disease of the Supra-renal Capsules. (Page 287.) From a drawing by Dr. Greenfield.

The drawing shows a section of part of one of the translucent greyish bands. Hartnack, oc. 3, object. 8.

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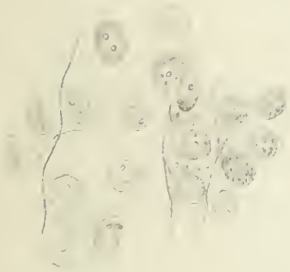

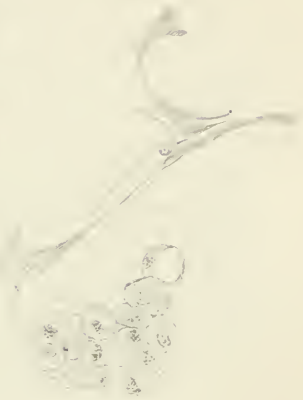

When some of these nuclei are separated by teasing out a specimen they are found to be surrounded by a small quantity of protoplasm irregular in shape and amount. Between the cells can be recognised a small quantity of fibrous stroma. On shaking a section in water a great part of the cells can be washed away, and it is then seen that the stroma takes an alveolar form, but even after the most prolonged washing it is found that some cells still remain embedded in the tissue of the stroma. The stroma is almost homogeneous in some parts, in others delicately fibrous. In many places small capillaries can be recognised in it, but they are not of unusual size or number. On examining an unwashed specimen with a higher power it is quite clear that the stroma penetrates in most parts between the individual cells. The nuclei are somewhat larger, as a rule, than white blood-corpuscles. The tumour is therefore a sarcoma and would perhaps be most properly classed as a small round-celled sarcoma. The alveolar appearance of its stroma is perhaps due in part to the more delicate intercellular substance being washed away with the cells. The small nodules in the medullary canal present the same structure as the external growth. The new bone surrounding the femur and filling the medullary canal seems to us rather the result of the irritation produced by the presence of the tumour than of ossification of the growth itself—1st, because of the great ease with which the tumour can be peeled off the new growth of bone; 2nd, because the new formation of bone extends considerably beyond the limits of the tumour; 3rd, because the surface of the bone beneath the tumour does not present the markedly spiculated appearance seen under ossifying sarcoma; and 4th, because the new bone shows signs here and there of being eroded by the growth of the tumour. A thin section from the surface of one of the small nodules of new bone shows nothing abnormal. The contents of the Haversian canals do not resemble the structure of the tumour. An examination of the growing surface of the tumour shows that it is implicating the surrounding parts like a true cancer. It is preceded by a small round-celled growth. This can be seen invading the muscles in many parts.

The specimen is interesting from its superficial resemblance to carcinoma, but very little examination serves to establish the difference. The cells are not of an epithelial type, but consist merely of irregular masses of protoplasm with a large nucleus in the middle. The stroma is much more intimately related to the cells than in

carcinoma, and is more delicate, being in many parts apparently almost membranous and devoid of structure. Again, in many parts the alveolar stroma disappears, and here the nature of the growth is quite evident. From the appearance of the growth and the way it is implicating the surrounding parts, we should fear it will prove malignant in its course.

HENRY ARNOTT.
MARCUS BECK.

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11. *An anomalous form of "blood-cyst."*

By RICKMAN J. GODLEE.

THE patient from whom this specimen was removed was a widow, æt. 67, who had never borne children, who had always enjoyed excellent health, and apparently had no hereditary tendency to malignant disease.

She first came under notice in January, 1873, with a tumour in the breast, which had been growing slowly for four months, and which was referred by the patient to a blow received on the breast about a year previously. It was diagnosed by Mr. Marshall, to whose kindness I am indebted for the opportunity of bringing the case forward, as a cyst, probably connected with the growth of a sarcoma, but as it was removed by the "flèche caustique" no opportunity was afforded for microscopical examination. In about three or four months recurrence took place in the cicatrix, accompanied this time by lancinating pains, and a year afterwards a small lump made its appearance in the axilla. The tumour at no time grew rapidly, and when she again presented herself at the hospital, though it had been growing for more than twelve months, its diameter was not more than 2 inches. It had very much the appearance of an ordinary scirrhus involving the skin. It was intensely hard, and the tissues were puckered round it as the result of contraction, while the skin over it was quite adherent and of a dark red colour. It was obvious, however,

DESCRIPTION OF PLATE XI.

Figs. 1 and 2 illustrate Mr. Godlee's specimen of Anomalous Form of Blood-cyst. (Page 270.) From drawings by himself.

FIG. 1. A section from the dense fibrous wall of the cyst.

A. Connective tissue forming an exceedingly coarse stroma, in which are seen at various parts cavities containing gland structure (c).

C, C.′ Epithelial structure arranged in convoluted tubes, cut in C more or less transversely, and at C′ more or less longitudinally. These tubes almost exactly resemble the acini of the breast of a puerperal woman.

B. Hæmorrhage which has taken place apparently into one of the tubes, for the cavity is lined in parts by epithelium, as is shown at *d*, *d*. An exaggeration of this formed the larger cyst. × 225.

2. Section of a gland in the axilla secondary to the blood-cyst of breast. This corresponds to C in fig. 1, more highly magnified.

Figs. 3, 4, 5, 6, 7, 8, and 9 illustrate Mr. Butlin's specimen of Warty Tumour growing in the interior of a Sebaceous Cyst. (Page 273.) From drawings by himself.

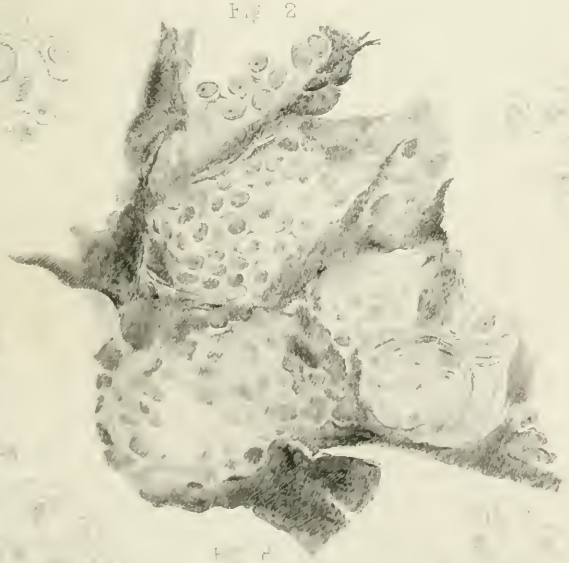
FIGS. 4, 5, 6, 7, 9. Forms of cell-nests.

3. Large nucleus with nucleolus.

8. Cell filled with nuclei. × 260.

Fig. 3

Fig. 2



on careful manipulation, that the mass really contained fluid. There was also the small lump mentioned above in the axilla. Mr. Marshall removed the two masses together with the knife, and it should be noticed that it was not easy to trace any direct communication between the tumour and the gland.

On making a vertical section of the tumour it was seen to consist of a dense fibrous capsule, about $\frac{1}{16}$ inch thick, which was closely incorporated with the tissues round about it, and which indeed sent fibrous prolongations into them resembling those seen in the neighbourhood of a scirrhous cancer. The cavity thus formed contained about two drachms of dark treacly blood, and besides this a considerable quantity of a semi-solid mass of dark brownish colour, which I have no doubt would have passed, some time ago, for modified blood-clot. The supposed gland in the axilla was made up of a similar capsule, divided, however, into two parts by a transverse septum, and containing only the gummous material, which in this case had a pinkish colour mottled with purplish spots, instead of the uniform brown which was seen in the larger cyst.

It was expected, from analogy with similar cases, that the soft material would turn out to be more or less modified sarcoma tissue, but on turning out a little of it in water it was found to consist of a number of tortuous tubes, which when unstained presented no appearance of structure, and were considered at the time to be enormously dilated capillaries; there were also a large number of cells of various shapes and sizes, and blood-corpuscles in more or less advanced decay.

It was very difficult to obtain a thin section of the gummous mass after hardening in spirit on account of its extreme brittleness; but when it was accomplished the tubes were found to be lined with a well-marked epithelium of very slight thickness and more or less hexagonal shape, with clear oval nuclei and minute nucleoli; the tubes were tolerably regularly circular in section, and held together by an almost imperceptible amount of connective tissue (*vide* Pl. XI, figs. 1 and 2). On comparing this with a section of the breast of a woman who had died a few days after confinement, it was found that the size of the tubes corresponded very nearly with that of the acini of the gland, while the characters of the epithelium were identical, the chief difference of the two being, in fact, in the much smaller amount of connective tissue and greater length and tortuosity of the tubes in the tumour. The amount of connective

tissue, however, in the puerperal breast is not great. The blood was partly in the tubes, partly in the vessels, and partly in cavities lined by epithelium, and thus presumably dilated acini.

To determine, if possible, the relation of the vessels to the tubes, some of the gummous material previously hardened was teased out, stained with logwood, and then mounted in Dammar. The extraordinary appearance was then shown of vessels of some size filled with blood-corpuscles, round which the twisted tubes were arranged in tolerably thick festoons. There were besides a number of capillaries which showed no relation to the tubes. On returning then to the section, here and there it was seen that similar systems of tubes and vessels had been cut transversely, so that the vessel was seen in the interior and the tubes were arranged round it with a slight clear interval between. A more careful examination of the section showed that the capillaries ran along the delicate septa between the tubes, which is an arrangement almost precisely similar to that described by Stricker as occurring in normal breasts. The microscopical appearances of the soft material in the large cyst and the axillary gland were the same.

The tumour, then, seems to present points of very considerable interest, both in regard to its relation to the group of cancers and its position among the "blood-cysts." I suppose it is pretty generally allowed that a cancer is a depraved growth of the gland in which it happens to be developed, the stroma answering to the interstitial tissue, and the cells to the gland tissue proper; but I do not think it often falls to our lot to see in an undoubted cancer, say of the breast, anything which approaches at all closely to normal gland structure. This growth, though to the naked eye it bears no resemblance to breast tissue, yet under the microscope it is almost indistinguishable from a normal breast under one of its own special functional conditions.

And then as regards its position amongst blood-cysts. I had the honour to report in the last volume of our 'Transactions,' vol. xxvi, p. 193, a case which differed in some important particulars from those hitherto recorded, and it was then stated that probably all blood-cysts which refuse to submit to ordinary methods of treatment are connected, in some way or another, with some form of sarcoma. This case illustrates the fact that a very similar condition may be produced by connection with a tumour which appears to find its place, not amongst the sarcomas, but amongst the cancers, and it

tends to show that, however useful the term "blood-cyst" may be as a clinical classification, as a pathological expression it is altogether valueless. *January 18th*, 1876.

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12. *Warty tumour growing in the interior of a sebaceous cyst (? papilloma).*

By Henry Trentham Butlin.

These portions of tumour were removed from beneath the scalp of a woman, æt. 48, who was admitted into St. Bartholomew's Hospital in January of the present year, under the care of Mr. Callender. The patient presented a tumour about the size of her fist, situated on the back of the head over the occipital and parietal bones. The skin covering it was perforated by ulcers; the base of the tumour was indurated, and was tolerably firmly adherent to the parts beneath. It had existed, according to the patient's statement, for many years, during the earlier part of which period it had altered very little; but during the last two years it had become much larger, hard, and ulcerated on the surface. Operation was performed in February. When the tumour was cut into it appeared to consist of a large cyst or sac, filled with these and similar growths. They shelled out quite easily from the interior of the cyst everywhere except at the base (*i. e.* over the skull), from which part they appear to have sprung. At this point they were so firmly adherent that they were with much difficulty removed; it was even feared that some portions of the growth were left behind. The wound, however, healed well, and there was no reappearance of the tumour when the patient left the hospital in March. When first they were removed the growths were much less firm, less coherent, more transparent, and of a much lighter colour than they have become since by the action of the spirit in which they have been kept. They looked at first like large masses of sago, or small rice grains which had been soaked in some fluid.

Microscopic examination of sections of the hardened masses showed them to possess the ordinary characters of papilloma or

epithelioma, it is not certain which, for the base from which or in which the tumour grew could not be obtained for examination. It was supposed, or rather hoped, that they might be papillomatous, since such tumours are more common in the interior of cysts than epitheliomata, and although there was induration about the base of the tumour, this might have been accounted for by its inflamed and ulcerated condition. Moreover, there was no projection of the growths through the scalp, and no glandular enlargement other than might easily have been accounted for by the irritable condition of the tumour when the patient came into the hospital. It seemed highly probable that the growths were produced in the interior of a sebaceous cyst, for they undoubtedly arose from the wall of a cyst of some kind. The position of the tumour, its duration of many years, and its tolerably large size throughout were all evidences in favour of this having been a sebaceous cyst.

This was one reason for my bringing the tumour before the notice of the Pathological Society. Looking through the 'Transactions' I could not find any quite similar case.

Another reason was the large number of cell-nests or laminated capsules found in all parts of the sections, and the early conditions of formation of most of these affording a good opportunity for the study of their development.

As far as I could discover there appeared to be two modes in which these nests were formed (*vide* Pl. XI, figs. 3 to 9). The first was described some years since by Thiersch, or perhaps even before his monograph on 'Epithelioma' was published. Multiplication by division of an epithelial cell taking place in the midst of a mass of epithelial cells, the surrounding cells are pressed outwards and flattened by pressure between the newly formed cells and the more distant cells. Fig. 7 seems to be an example of this form of cell-nest; fig. 4 perhaps an earlier condition of the same process in which division of the nucleus and enlargement of the cell are the chief features.

The second mode is analogous to, if not the same as, that described within the last few weeks by Dr. Gowers in a paper on the Nest-celled Sarcomata read before the Medico-Chirurgical Society. One cell is gradually formed within another, in what manner does not certainly appear. As it grows it presses out the protoplasm of the mother-cell until this assumes the form of a thin ring or crescent, in some part of which is generally seen its nucleus. This process,

repeated in the secondary cell, may be again and again repeated with the result of producing a smaller or larger nest. The secondary cell is rarely placed in the centre of the mother-cell, so that the forms produced are most commonly those of semilunes or crescents. Examples of these conditions are shown in figs. 6 and 9. Although vacuoles are by no means uncommon in the epithelial cells, I was not able to discover the growth of secondary cells within those vacuoles. Fig. 3 shows a second nucleus in a cell increasing to a considerable size, and containing two very large nucleoli. It may be that the secondary cell is formed in this manner, the resemblance of this nucleus to many of the cells being further strengthened by the facts that its borders were finely dentated, as was frequently the case with the cells (riff-zellen).

There was not anywhere any appearance of connection between the cell-nests and gland ducts or acini. *May 16th*, 1876.

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13. *Lymphadenoma, with infiltration of the lungs and skin.*

By W. S. Greenfield, M.D.

Eliza K—, æt. 45, was admitted to St. Thomas's Hospital, on September 30th, 1875, under the care of Dr. Peacock (to whom I am indebted for the clinical history and the opportunity of bringing the case under the notice of the Society).

Family history.—Father living, æt. 67; mother died of "dropsy" at the age of 42; one brother and a sister are living; one brother died in infancy.

Previous history.—The patient has been subject to rheumatism and had an attack of rheumatic fever three months ago. She has also been subject to cough for some years.

For some time past she has been suffering from weakness and loss of flesh, and some months ago she noticed some lumps in the groin. About three months ago she also observed some swellings in the skin. She has, however, had no urgent symptoms until three weeks before admission, since which time she has suffered from sore throat and cough.

On admission.—She is pale and somewhat emaciated; the lips are

purple, but she does not seem to have any material difficulty in breathing; she has a hacking cough, but only expectorates scanty sputum with dark spots in it. She sweats profusely; appetite good; bowels open. There is considerable enlargement and induration of the cervical glands on the right side. On the skin of the chest wall, both in front and behind, there are several small rounded tumours, flattened on the surface; some of small size, situated beneath the skin, which is not discoloured over them; others larger, fully the size of a florin, and involve the skin, the surface being generally reddened, and with small varicose vessels ramifying over them. They are very hard and somewhat tender to the touch. In addition to these there are numerous enlarged glands on each side of the neck and in each groin. One large nodule is more deeply seated beneath the right false ribs.

There is some deformity of the chest, due to the prominence of the middle part of the sternum and of the ribs attached to it, and some bulging in the left scapular region. No impairment of resonance in front; but expiration prolonged. Heart sounds normal; liver and spleen not enlarged; hepatic dulness 4½ inches in right nipple line; urine not albuminous.

October 23rd.—Glands in both groins still enlarged; veins of the forearms and over the front of the chest dilated and prominent. Temperature 98·8°; pulse 100; respiration 44; some dyspnœa. Front of chest normally resonant, posteriorly the upper half of the left chest absolutely dull, and so also over the upper part of the infra-spinous region on the right side. Breath sounds here very harsh; elsewhere dry and normal. Uterus normal.

November 3rd.—Profuse perspiration; no shivering; spleen somewhat enlarged. Temperature last evening 99·6°; pulse 120; respiration 48.

7th.—Complete loss of appetite; occasional vomiting.

11th.—Right lobe less resonant than left; less air heard to enter, and respiratory sounds higher pitched; vocal resonance much diminished.

15th.—Crepitation at right base posteriorly, with very little respiratory sound.

23rd.—Has continued in much the same state, suffering from bronchitis and consequent dyspnœa. Is unable to take solid food, but takes other diet well; tongue nearly clean. Pulse small and feeble, but not quick. Percussion note less clear on right than left

side all over; and both inspiratory and expiratory sounds are short and imperfect, on the left side attended with decided stridor; respiration is everywhere attended with sibilant and sonorous rhonchus. There are numerous enlarged glands in the neck, but probably not larger than before.

During the month of November the temperature varied from about 100° to 102·6° F., with no constancy as to the occurrence of greater elevation in morning or evening.

No further notes were taken, the patient gradually growing weaker and becoming more emaciated; she died on January 28th, 1876, at 5.15 a.m. It was, however, observed that the nodules in the skin diminished considerably, and some of them became almost imperceptible.

Post-mortem, January 29th, thirty hours after death.—Body much emaciated; anasarca of both lower extremities, not extending above the knees.

On the upper and inner part of the left thigh, occupying the greater part of Scarpa's triangle, was a hard mass, very little if at all elevated above the surrounding surface, but feeling like a brawny induration of the skin, with the subcutaneous tissue adherent to it; some harder nodulation here and there. The skin over this patch was of darker colour than elsewhere, the discoloration being produced by a pigmentation which appeared to be solely around the hair-follicles. On making an incision into the tissues the skin was found to be considerably thickened and firmly adherent to the subcutaneous fatty tissue, and the fat, connective tissue, and lymphatic glands in the space were fused together into a hard mass, which appeared to consist of a chronic inflammatory thickening of the whole of these tissues, with enlargement of the lymphatic glands. The femoral vessels passed through and were surrounded by this mass, but there did not appear to be any infiltration of their walls.

The *lymphatic glands* in the left groin were greatly enlarged, and formed a mass which was continuous upwards beneath Poupart's ligament from that in Scarpa's triangle. The glands along the course of the left external iliac vessels were also greatly enlarged, several of them of the size of walnuts; for the most part softish, and white on section, but in some parts of a greyish or slaty colour on the surface (? discoloured by adjacent intestine). Some of the inguinal glands were also discoloured in places. The

glands in the right groin were also enlarged and soft. So also were those along the right external iliac vessels, though much less so than on the left side. These latter glands were firm, of dark colour, and on section of an uniform blue blackish-grey colour throughout.

The lumbar glands were enormously enlarged, forming irregular nodulated masses in front of the spine, one large mass partially surrounding the pancreas and another lying in front of it. The large masses were formed by the coalescence of swollen glands, with but little or no intervening fibrous tissue; and several of the separate glands were of the size of large walnuts. The mesenteric glands were also greatly enlarged and produced large tumours by their confluence. The glands were for the most part pale, of yellowish-white colour on section, softish, not exuding any creamy juice on scraping. Some of them showed slight greyish discoloration here and there. Some of the mesenteric glands were, however, quite black, firm, and not enlarged.

The glands in the anterior mediastinum were of almost normal size, only one or two being of the size of a hazel nut. In the posterior mediastinum all the glands were somewhat but not greatly enlarged, with the exception of one or two in the bifurcation of the trachea, which formed a large mass, and one the size of a large walnut, which lay immediately above and in contact with the left bronchus, which was thus enclosed between the two.

In both axillæ, but chiefly in the left, the glands were generally enlarged but not confluent, being for the most part of deep purple colour on the surface, and on section presenting a mottling of deep purple colour surrounding small yellowish-white points; the darker colour being apparently due to intense inflammatory congestion. The glands were soft and succulent. The glands in the neck showed but slight traces of enlargement.

In the *skin*, chiefly over the thorax, were several patches of rounded shape, flattened, scarcely elevated above the surrounding skin, which from the surface, felt like tumours in the subcutaneous tissue adherent to the skin. They corresponded in situation with those observed during life, but had lost all trace of vascularity, and had not the aspect of tumours as seen from the exterior. Some of them indeed appeared to be merely local indurations of the skin. On cutting into them they appeared to consist of a thickening of the subcutaneous fatty tissue, with hypertrophy of the superjacent skin, the structures to the naked eye looking in all other respects

normal, presenting none of the features of new growth. There was no sign of lymphatic glands beneath them, nor were they adherent to the subjacent tissues.

On examining sections from the simply thickened portions of skin (*i. e.* without deep infiltration of the subcutaneous fatty tissue) under a low power of the microscope, after hardening in Müller's fluid, the whole of the structures appeared to have undergone some swelling and to be irregularly infiltrated by a small-celled growth. In the subcutaneous fatty tissue it appeared as an uniform mass of small cells in which a few rounded spaces, the remains of vessels and of fat-cells, persisted. The growth extended continuously into the deeper layers of the corium, the fibrous bands of which were separated by masses of cell growth. Smaller patches and bands of the new growth were also scattered irregularly throughout the corium, especially in the neighbourhood of and surrounding the sweat-glands and hair-follicles, reaching to immediately beneath the rete Malpighii in some places. The rete also appeared somewhat thickened.

With a higher power the new growth appeared to consist almost entirely of small rounded cells, densely aggregated together, with a small quantity of intercellular substance, no distinct stroma being at first sight seen, but in brushed sections and at the margins a very distinct branching stroma formed by branching cells and protoplasmic filaments was discovered, the tissue resembling in every respect the so-called adenoid tissue. The condition of the skin covering the mass of enlarged glands in the groin was precisely similar, the stroma of the new infiltrating growth being in some parts more dense and fibrous (*vide* Pl. III, figs. 4 and 5.)

Pleura.—Right, with slight recent inflammation all over the surface of the lower lobe, which was roughened and covered by a thin layer of recent lymph; the diaphragmatic surface adherent by a firm layer half a line thick. Left normal.

Lungs.—Right weighed 27 oz., its lower lobe was nearly solid throughout. On the surface were several deeply depressed and puckered scar-like patches, without any thickening of the pleura covering them. The substance of the lung, but especially of the lower lobe, very hard to the touch, and irregularly nodulated. At the root of the lung the vessels and bronchi were surrounded by a mass of infiltrating new growth, the walls of the bronchi being greatly thickened, and measuring from 3 to 6 millimètres. The

branch of the pulmonary artery to the lower lobe was much thickened and flattened by compression; its lumen was occupied by a firmly adherent decolorised thrombus. The thickening of the walls of the bronchi and vessels extended for some distance into the lung.

On section of the lower lobe the consolidation appeared to consist of a general infiltration and induration of the tissue, with, in addition, numerous nodules of various size, which were for the most part continuous with the infiltration which extended from the root of the lung along the bronchi and vessels. The walls of the latter, especially the bronchi, were much thickened, of white colour, which was more marked towards the root of the lung, the growth appearing to be mainly in their outer coat or connective tissue sheath. The greater part of the induration of the lung tissue proper was of an iron-grey colour marbled with black, closely resembling Purbeck marble in colour and appearance, of uniformly hard consistence throughout, and showing no sign of softening in any part. The nodules were well-marked, though not very sharply outlined, slightly prominent on the cut surface.

The mucous membrane of the bronchi was somewhat swollen and congested.

The middle lobe was also almost uniformly consolidated, the upper for the most part crepitant.

The left lung weighed 20 oz., its lower lobe and the upper part of the upper lobe adjacent to the root nearly solid, and presenting similar characters with the right, but the infiltration less nodular and more generally diffused. The upper lobe was for the most part crepitant.

The bronchial glands at the root of the lungs were involved in, and had apparently furnished the starting-point of, the masses of growth in that situation.

On microscopic examination, scarcely any normal lung tissue could be seen in the affected parts, the whole being infiltrated with new growth. This consisted of adenoid tissue resembling that in the skin, but in a much more advanced state of growth. In many parts the stroma largely preponderated, and was of a very dense fibrous character; resembling in structure an indurated lymphatic gland, or the lung tissue in chronic phthisis. It appeared to radiate from the walls of the bronchi, around which it was of dense fibroid character, into the walls of the air-cells, where in its earlier stages it appeared as mainly a growth composed of small round cells with

scanty stroma. The alveoli, where least affected, presented evidences of acute pneumonia.

Heart, small; normal; the lymphatics on the surface extremely well marked out.

Spleen of enormous size, reaching five inches below the ribs in the left nipple line, extending to the middle line in front. It weighed 63 ounces, measuring vertically ten inches, transversely seven inches, and was two and a half inches thick at its thickest part. It was of somewhat oblong shape, and of deep purple colour on the surface, on section of uniform deep purplish-chocolate colour, no sign of the Malpighian corpuscles remaining; the tissue very soft and friable, pulp readily washed away, and the stroma scanty. The appearance was that of extreme congestion and proliferation of the splenic pulp, without any corresponding increase in the trabecular tissue.

The microscopic characters of the enlargement of the organ were extremely difficult to make out, its extreme softness rendering it impossible to make a fair section, even after a long course of hardening in various reagents. But its tissue appeared to consist almost entirely of a mass of small cells, varying from $\frac{1}{1000}$ to $\frac{1}{2000}$ inch in diameter, roughly speaking; for the most part rounded, and with a distinct nucleus and nucleolus; some, however, were elongated and fusiform and others branched. The stroma was greatly diminished in relative quantity, no sign, in fact, of the normal structure being discovered on the sections examined. It may be added that the cells presented, for the most part, no marked difference from those of the normal splenic pulp.

Liver pale, somewhat atrophied, otherwise normal, weighing 53 oz.

Kidneys pale, firm, normal; weight 10 oz.

Pharynx normal.

Œsophagus.—Some infiltration of the submucous tissue at the upper part for about 2 inches, with a substance resembling tubercular growth, producing a slight irregular nodulation of yellowish colour. One small shallow oval ulcer $\frac{1}{3}$ inch in length was found here.

Other organs normal.

Remarks.—So far as I have been able to ascertain, the case here related is peculiar in the fact of the occurrence of growths of lymphadenoma in the skin, apart from infiltration by continuity with lymphatic glands. The majority of the tumours in the skin were dis-

tinctly out of the region of the lymphatic glands generally recognised and known to exist, and, moreover, sections of them when examined microscopically proved that they consisted of infiltration of fat where they involved the subcutaneous tissue, no trace of a gland being discovered, as would probably be the case had they started in small glands. That they started in lymphatic tissue is very probable, if not capable of demonstration. The very marked diminution in size of the nodules, in some cases amounting almost to disappearance to the naked eye, except when a section of the skin was made, was probably due to the gradual absorption of the infiltrated fat, which at first would be only infiltrated and increased in bulk.

The occurrence of these tumours in the skin gave to the case clinically the character rather of sarcoma or cancer than of lymphadenoma. In only a small proportion of cases of cancer, it is true, do these cutaneous tumours occur, except in some cases of cancer, particularly of the breast,where a sort of local infection of the skin in the neighbourhood of the tumour leads to the production of numerous small nodules in it. But in rare cases a general growth of small nodules in the subcutaneous tissue, involving the skin secondarily, occurs apart from the contiguity of a mass of new growth. Secondary sarcomatous nodules in the skin are, I believe, of more frequent occurrence, and in those which have come under my own observation during life have very closely resembled, in their external features, those found in the present case. In spite of this peculiarity the very general glandular enlargement and other features of the case led Dr. Peacock to form the correct diagnosis of lymphadenoma.

As regards the infiltration of the lung the case is not by any means unique, although the characters and distribution were somewhat peculiar. Dr. Dickinson (see 'Path. Trans.,' vol. xxiv) has recorded a case in which there was a mass of lymphomatous growth lying in front of the trachea, from which a thick diffused induration extended around the bifurcation of the trachea and along the bronchi and blood-vessels, entering the root of the right lung, and as thick plates following the laminæ of conective tissue, penetrating as thick sheaths for some inches, and causing partial consolidation of the lung. In this case there was no general enlargement of the lymphatic glands of the body. Dr. Moxon also mentions a similar case of local infiltration of the lung.

It seems to me a matter of some doubt whether this case is to be regarded as one of 'Hodgkin's disease.' Certainly the symptoms

were of very different character from those in typical cases of that disease, the name of which is now far more strictly applied than it was when Dr. Wilks first crystallised the experience of Dr. Hodgkin and himself in that clinical term. In two or three respects more particularly does this case differ from them. The glandular affection, although very widespread, was not by any means general. There was a very marked absence of affection of the cervical and some of the mesenteric glands, and those in the right groin were comparatively slightly affected. From the fact of the very great affection of the glands in the left groin, where a firm indurated mass involving skin, fascia, glands, and the sheaths of the vessels existed, this would seem to have been the starting-point of the disease; from this point the infection appeared to have extended to the left iliac and the lumbar glands, and thence upwards to those of the posterior mediastinum, thence by the bronchial glands to the root of the lung and along the lymphatic trunks into the lungs. The entire absence of affection of the glands in the anterior mediastinum and the relatively slight implication of the cervical and axillary glands (that of the latter being evidently in an early stage) also tend to confirm this view of the course of the disease. Now, had the growth been a sarcoma or cancer originating in the same region, viz. about Scarpa's triangle on the left side, the course of infection might have been identical, and the *post-mortem* appearances nearly the same, modified, doubtless, by the peculiarly rapid and exuberant growth which lymphoma affects. Cases might readily be cited in proof of these views, but the facts are well recognised. It may be urged that in typical cases of Hodgkin's disease the affection of the glands is not by any means symmetrically equal, and that the extent of implication of other organs presents a great variety, both in situation and amount; but it is, I think, rare to find so definite a course of infection from one set of glands to another. Indeed, one of the most marked features of the typical cases is the very rapid, almost sudden, enlargement of glands in various parts of the body, accompanied by or following a febrile attack, the enlargement often subsiding to a certain extent, and then recurring with progressive increase from time to time.

Again, the peculiar anæmia, and what I am inclined to regard as even more characteristic, the recurrent febrile attacks, were in this case but little prominent, if they existed. There was, it is true, some elevation of temperature from time to time during November,

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Again, the peculiar anæmia, and what I am inclined to regard as even more characteristic, the recurrent febrile attacks, were in this case but little prominent, if they existed. There was, it is true, some elevation of temperature from time to time during November,

but fever was never a marked symptom until the fatal attack of pneumonia.

Again, the condition of the spleen was quite different from that usually seen in Hodgkin's disease, in the fact that the sole morbid change seemed to consist in proliferation of the pulp, instead of a growth of lymphatic tissue in the form of small nodules, which Dr. Wilks has aptly compared as regards their naked-eye characters to lumps of suet. The enlargement seems to have been rapid, and to have occurred late in the disease.

It would be out of place here to discuss the general question of the relations of "lymphadenoma" with "Hodgkin's disease," or to inquire what limit is to be given to the latter term. There is a large class of cases of lymphadenoma in respect of which no question can arise; they have none of the features of that disease. Other cases present all the clinical characters, together with the pathological facts of glandular affection and secondary implication of other organs. But there is a large class of cases, the number of which on record is gradually increasing, in which general enlargement of the glands and spleen and the growths in other organs occur, but without any of the clinical characteristics of "Hodgkin's disease." It is mainly as a contribution to the final determination of this problem that the present case is brought forward.

An able review by Dr. Murchison of most of the cases recorded in the 'Transactions' will be found in vol. xxi (1870), p. 194; and a comparison of this with some remarks by Dr. Moxon, in vol. xxiv, and with Dr. Wilks's original paper, will, I think, show that there is a good deal of uncertainty on the questions here raised.

May 16th, 1876.

IX.—DISEASES, ETC., OF THE DUCTLESS GLANDS.

(A) DISEASES OF SPLEEN AND LYMPHATIC GLANDS.

1. *A portion of the spleen from a case of general tuberculosis.*

By F. Charlewood Turner, M.D.

The specimen exhibited was taken from the body of a patient of Dr. Peacock's in St. Thomas's Hospital. By his kind permission I am able to bring it under the notice of the Society.

It is part of the spleen of a boy, H. F—, æt. 11, who was admitted into St. Thomas's Hospital, on February 2nd, 1876, with symptoms referable to an irregular attack of enteric fever, of which there was a history of possible contagion from a case occurring in the same street.

The patient, though always thin and delicate, had enjoyed good health until a month previously, when his illness commenced with shivering and pains in the limbs and abdomen. He had continued ill up to his admission. He then was much emaciated, and had moderate fever, his temperature varying from 99° to between 101° and 102°, and subsequently rising as high as 103°. There were no abdominal symptoms, with the exception of a considerable enlargement of the spleen, the edge of which could be felt; the tongue was red at the tip and edges, and the bowels were confined. With the exception of gradually increasing emaciation, and the occurrence of slight rhonchus in the chest, and latterly an excessive degree of fretfulness and irritability, his condition continued without material change until four days before his death, when the nature of the case became evident. On the evening of March 8th he became delirious, and was subsequently conscious only at intervals; on the evening of the 12th he became comatose, with rigidity of the limbs and strabismus, and early on the following morning he died.

There was no history of any tendency to phthisis in the family.

At the *post-mortem* examination the appearance of the organ, exhibited, was very remarkable. It was seen to be much enlarged, weighing fifteen ounces; it was of a dark colour from venous congestion, though mottled throughout, and rendered uneven by whitish nodular projections of circular outline, and about the size of peas, at the summit of some of which were perforations of the capsule, and of the layer of lymph which covered it throughout, permitting the escape of softened-down caseous matter from within, into the peritoneal cavity. This appearance of the cut surface of the fresh organ presented a still more striking contrast between the blackly congested spleen tissue and the tubercular masses scattered through it—an appearance recalling that of the "hard-bake" spleen, except that the white nodules were smaller in size, and were also all softening down in the centre; in some parts these had run together, forming larger, irregular excavations.

Tubercles, apparently of a later date, were disseminated through the lungs, liver, kidneys, and supra-renal capsules, none of these were found softened down.

There was a mass of enlarged lumbar glands caseous and softening down, and forming a cavity of the size of an egg. This seemed to be the seat of the oldest disease.

The under surface of the diaphragm in contact with the liver was covered with tubercles. There was a small quantity of serous fluid in the abdominal cavity.

There were two small superficial ulcers in the ilium, close to the ilio-cæcal valve.

The brain presented the ordinary appearance of tubercular meningitis. *May 16th*, 1876.

(B) DISEASES OF SUPRA-RENAL CAPSULES.

2. *Supra-renal capsular disease, with bronzing of the skin.*

By Thomas B. Peacock, M.D.

C. M. H—, æt, 34, a married woman without any children, and who had never been in the family way, was admitted into Alice Ward, St. Thomas's Hospital, on the 5th of August, 1875.

Her father is living, but her mother died at the birth of her second child, the patient's only sister. She was supposed to have been phthisical previously. Her father is of dark complexion, and her mother herself was particularly sallow; and she has also always been of a very dark complexion and has dark brown hair.

Seven years before her admission into the hospital she was out of health, suffering from debility, and two years before she had eczema, which affected the face, arms, and feet, and she was then much sallower than before. For this she was treated by Mr. Hutchinson, at the Hospital for Diseases of the Skin, Stamford Street, and got well in about two months, though the skin never recovered the natural colour.

She had always been short-breathed and had a cough with expectoration, but had never spat blood. Her appetite was very variable; she would sometimes be quite ravenous, and at others could scarcely take any food. Her digestion was defective, and the bowels were generally confined. She was always most abstemious in her habits, rarely ever tasting any kind of stimulating beverage.

About the beginning of May she was sent to Dr. Peacock for his opinion as to whether she was in a state of health to admit of her going as an emigrant to New Zealand. The darkness of her complexion was then very striking, and she complained of dyspeptic symptoms, and of cough, expectoration, and shortness of breath, but no signs of disease were detected on examining the chest.

Since she had been seen by Dr. Peacock she had been getting increasingly out of health, becoming more feeble and languid; and latterly she had had frequent attacks of faintness, in which, how-

ever, she has never entirely lost her consciousness. The catamenia had been absent for two months.

For three weeks before her admission she had been confined to bed, had had constant sickness and vomiting, so that she was scarcely able to retain anything which she took, and she was much troubled with hiccup and retching. Her complexion had become rather lighter recently.

When admitted into the hospital the symptoms of supra-renal capsular disease and the darkness of the skin were very marked. The bronzing was particularly distinct on the forehead and face, the backs of the arms and hands, and in the folds of the axilla and of the elbows. There were two or three very dark patches on the dorsum of the tongue, which contrasted strikingly with the paleness of the mucous membrane generally. On the evening of her admission into the hospital, and the following morning she had a temperature of 100·8°, but with these exceptions her temperature ranged between 98·8° and 98·4°.

While under observation she had no active symptoms, but simply complained of excessive weakness and inability to take food. She died quite suddenly, having apparently fainted and being slightly convulsed, on the evening of October 9th.

Post-mortem, five hours after death. By Dr. Greenfield.

Body much emaciated. Very marked bronzing of the skin of the face and neck, of the hands and forearms to just above the elbows, also of the axillæ and flexures of all the joints, the areolæ and the perinæum. The pigmentation is much more marked on the forearms than is usually the case, as compared with the general colour of the trunk, and has an appearance similar to that produced by constant exposure of the arms as high as the elbows.

On the edges and inner surface of the lips and on the buccal mucous membrane there is also very marked pigmentation in small scattered patches, which on close inspection appear to be made up of minute black or purplish dots. Along the dorsum of the tongue, near to but not actually at the edges, are several small patches of pigmentation of inky colour, forming an irregular band along each side of the dorsum ; on the under surface and the tip are also several well-marked patches. The pigment is mainly localised around the papillæ, which are not themselves involved.

Thorax.—Pleura normal.

Lungs, both extremely pale in colour, almost bloodless; tissue crepitant, very emphysematous; the right weighing only ten ounces, the left eleven and a half ounces. They pit deeply on pressure, and have much the appearance of those after death from hæmorrhage. Bronchi normal. On section the tissue for the most part crepitant, one or two small scattered patches of recent lobular pneumonia being found in the lower lobes, and a few small pigmented fibroid cicatrices, none larger than a sweet pea. In the upper lobe of the right lung are one or two small calcified nodules surrounded by deeply pigmented fibrous tissue. The greater part of both lungs, however, is entirely free from traces of old or recent inflammation. Bronchial glands normal.

Perieardium normal. *Heart* weighs five and a half ounces, very small; left ventricle firmly contracted and empty, right containing a small quantity of dark fluid blood; valves healthy. The muscular tissue is of dark brownish colour, somewhat soft, but free from signs of fatty degeneration.

Peritoneum.—Very numerous bands of tough fibrous adhesion connect the liver and spleen with the diaphragm and the neighbouring viscera, these adhesions being remarkably tough and firm on the under surface of the liver, where there is also some general thickening of the capsule of that organ.

The *liver* weighs 38½ ounces, is rather small and flabby; on section the tissue pale and apparently in a condition of simple atrophy.

Pancreas normal. *Spleen* weighing 7¾ ounces, of large size, rather firm, and of dark colour on the surface; on section the Malpighian corpuscles are very distinct, the pulp firm and of dark purplish colour.

Kidneys weigh together 8 ounces; capsule slightly adherent and tearing the cortex on separation; substance firm; cortical substance pale, somewhat wasted, but free from other sign of disease and entirely devoid of congestion.

Supra-renal capsules.—The right capsule is firmly adherent to the under surface of the liver, and is surrounded by a mass of dense fibrous tissue. The left is also surrounded by a dense fibrous investment, and attached by tough fibrous bands to the surrounding parts, some of these bands having small, pearly, bead-like nodules along their course.

The *right* capsule is enlarged, of somewhat oval shape, elongated

in a vertical direction, and is of pretty uniform thickness throughout. The surface is slightly irregular and nodulated, the organ very dense and firm.

The *left* is smaller than the right, and of more irregular shape; thickest in its lower half, where it measures five eighths of an inch in thickness. On section it presents a very characteristic appearance, consisting in great measure of a semi-caseous material resembling the section of a horse chestnut or of a syphilitic gumma, of yellowish-white colour, smooth, firm, and slightly shining on the cut surface, the whole being surrounded by an investment of rosy greyish fibrous tissue, from which bands pass inwards and traverse the caseous material in various directions, forming septa enclosing caseous nodules.

On microscopic examination of sections from the left capsule, after hardening in Müller's fluid under a low power, the structure appears to consists of granular masses surrounded by a nucleated fibroid growth. On examining with a higher power the granular-looking masses are found to be composed of amorphous substance with some granules of fat and specks of highly refractile material, the appearances being those of calcifying caseous foci. The fibrous septa consist in some parts of thick fibrous bundles closely resembling ordinary areolar tissue, with small round cells running between and around the fibres. In other parts they are mainly composed of dense aggregations of small round cells resembling "lymphoid" corpuscles in all respects, with only a scanty fibrous stroma; but for the most part they are formed by a reticulated fibrous stroma with coarse fibrillar bundles enclosing elongated spaces in which are contained similar small round cells. The latter closely resemble the tissue from a chronically indurated lymphatic gland (*vide* Pl. X, fig. 7).

The *retro-peritoneal lymphatic glands* are greatly enlarged, forming by their coalescence a hard nodulated mass along the spine, more especially on the left side. Those in the neighbourhood of the solar plexus are especially enlarged and hard. On section they are for the most part caseous and contain numerous cretaceous nodules. Some of them are acutely swollen and very vascular.

The other organs present nothing abnormal.

October 19th, 1875.

(c) DISEASES OF THYROID AND THYMUS GLANDS.

3. *Enlargement of the thyroid gland, principally of the right lobe, displacing the trachea and interfering with its form.*

By LENNOX BROWNE.

ELIZABETH C—, æt. 38, married, but without children, and residing at Luton, in Bedfordshire, a goitrous district, applied (December 15th, 1875) at the Central London Throat and Ear Hospital on account of shortness of breath caused by a swelling in the neck.

Her *history* was that fifteen years previously she had first noticed a small lump in the centre of the throat at the situation of the thyroid gland. As it increased it seemed to move to the right side, and for the last four months a similar small tumour has shown itself on the left side. No pain or inconvenience was experienced until twelve months ago, when the breathing was first affected, becoming short, not only on exertion, but even when sitting still; on talking the voice quickly became fatigued, "as if for want of breath." She found herself unable to lie down, and was obliged to sleep sitting up with the knees bent. Lately she has had great pain in the right shoulder, and cannot even bear the weight of the bedclothes on that side.

For eight months past she has noticed that the right side of the face does not flush or perspire. The right side of the face when hot being pale and dry, while the left is flushed, with the perspiration standing out like beads. The line of demarcation is distinct, and in the median line. There is no moisture in the right armpit, whilst there is in the left. Perspiration is normal at right elbow and palm. During this same period she has had marked ptosis of the right eyelid.

She is in fair general health, her appetite is good, but she is weak and rather emaciated, and very restless in her sleep. She has a troublesome hacking cough, with clear expectoration like simple saliva, but rather more adhesive. Has rather a difficulty in expectorating, but is always relieved after so doing. She has occasional,

but not frequent, attacks of a suffocative character occurring during the night. The voice is somewhat hoarse, weak, and laboured. Has frequent occipital headache.

Menstruation excessive in frequency, amount, and duration, for the last six years.

Both the father and mother had died of consumption. She has but two sisters, both of whom are married, with families in good health, and without any tendency to thyroid enlargement.

On examining the patient there is seen a general enlargement of the neck in the region of the thyroid gland. The right lobe is as large as a hen's egg, hard in texture, pressing back the vessels of the neck and pushing the trachea considerably towards the left side. The enlargement, which is of the fibrous variety of bronchocele, does not appear to interfere with the gullet in any way. The right side of the face and neck are quite pale and dry, while the corresponding parts of the left side are rather over-flushed and moist.

With the *laryngoscope* the right inner wall of the trachea immediately below the vocal cords is seen to be pushed in, so that the calibre of the canal is considerably diminished. The trachea is also displaced, but there is nevertheless ample room for the passage of air.

The *temperature* of the left axilla is one degree higher than normal, that of the right being normal. The pulse is 100 and weak. Dr. Fancourt Barnes has kindly made a sphygmographic tracing, which shows increased tension of the right radial artery, and also that the secondary waves are more pronounced on that side than on the left.

The right pupil is contracted and but little acted on by light. Both optic discs are decidedly and equally anæmic, but not depressed. Calibre of the vessels is normal, and the fundus generally is illuminated with difficulty. Reads Snelling No. 1 by artificial light equally well with both eyes (Mr. Bowater Vernon).

The case is interesting in connection with others previously reported by the author at this Society,[1] as illustrating how displacement of the trachea may be considerable without interference with respiration, but how the least interference with the calibre of the windpipe will at once embarrass the breathing. The disturbance of the sympathetic nerve is also noteworthy, and it

[1] 'Transactions' of the Society, vol. xxv, p. 255.

will be interesting to note the effect of local treatment on this symptom.

August, 1876.—A seton was passed through the tumour on January 4th, and retained five months. The breathing was very quickly relieved, as were all the other symptoms except those of a nervous character. At this date the ptosis is diminished; the right pupil acts better; perspiration is free in the right armpit and neck, but not on the right side of the face; the temperature is normal, and the arterial tension of the right radial is diminished. The tumour is very greatly reduced, the neck measuring barely 12½ inches, and with the laryngoscope the trachea is seen to be normal in direction and in its circumferential capacity and uniformity. The patient sleeps well, and has gained flesh.

December 21*st*, 1875.

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4. *Larynx and trachea three years and nine months after thyrotomy.*

By W. Pugin Thornton.

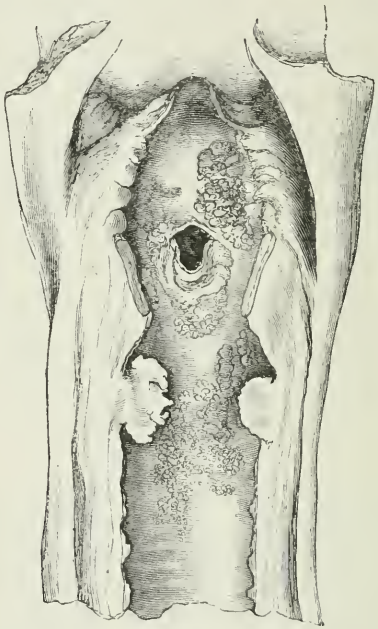
On June the 18th, 1872, I performed thyrotomy on Alfred G—,[1] a boy, aged two and a half years, for the removal of laryngeal growths. Six months after, recurrence of the growths having taken place, it was necessary to again open his windpipe, and from that time to the day of his death the child wore a tracheotomy tube. Once or twice every month during the first year following the performance of tracheotomy growths had to be removed from the tracheal opening, but after that period they gradually ceased to grow in that position, and for the year previous to his death the wound kept perfectly patent. The child had since the operation spoken in a loud gruff whisper. He died March 31st, 1876, of general marasmus following hooping-cough, which had attacked other members of the family.

The specimen showed numerous warty growths in the larynx, with a few scattered vegetations in the upper part of the trachea (*vide*

[1] This case is fully reported in the Clinical Society's 'Transactions,' vol. vi, p. 92.

Woodcut 7). The mucous membrane of the posterior wall of the trachea, where the end of the canula came, was thickened and surmounted with small growths. It was seen that none of the cartilages (thyroid, cricoid, and two upper tracheal rings) divided in the operation of

Woodcut 7.



thyrotomy had reunited, but that they were joined together by fibrous tissue; also that the canula had occupied a space between the cut portions of the cricoid cartilage and the crico-thyroid membrane. At the operation the tube was inserted between the two upper tracheal rings, therefore it must have worked its way into the position described. *May 16th*, 1876.

X.—DISEASES, ETC., OF THE SKIN.

1. *On some cases in which molluscum contagiosum occurred as a general eruption over the body and limbs of adults.*

By Jonathan Hutchinson.

We are chiefly familiar with molluscum contagiosum as an eruption on the faces of children, or less frequently on the necks, faces, or breasts of adults. We see it also now and then on the genitals of adults, when it is sometimes mistaken for venereal sores, and now and then around the nipples of nursing mothers, when, again, if solitary, its hard base may lead to an erroneous diagnosis of chancre. It is, however, very seldom either in children or adults that we see it as a general eruption covering the whole body. The object of my present communication to the Society is to describe several such cases, one of which presented also other very peculiar features in respect to the very small size of the individual spots. I am not aware that any such cases have as yet been described by authors. Hebra's two portraits of molluscum contagiosum are both of them from adults, and one of them shows the neck of a woman sprinkled over with the spots, many of them small, and not dissimilar from those which were present in my case, but the eruption does not appear to have been general, whilst in the second the penis is covered with spots, but again the eruption, though copious, appears to be local.

The following notes are those of the case to which I have alluded, and in illustration of which I exhibit to the Society a life-size portrait by Burgess.

Case I.—John T. L—, æt. 56, a gouty subject and inheriting the arthritic diathesis, was sent to me by Mr. Tay on account of a copious eruption of molluscum spots on the trunk, shoulders, and arms. On the back, breast, and shoulders many of them were as large as split peas and in all stages of progressive ejection from

the skin; almost all were a little constricted at their bases, and some were quite pedunculated; many showed a small central depression. On the arms, where the eruption was very copious, it showed some peculiarities; the spots here were very small, none being larger than No. 6 shot; most of them were pointed, and they were so smooth and semitransparent that they looked just like vesicles. On careful inspection I found minute horns projecting from many of them, and these, when pulled out with forceps, proved to be little plugs of very firm sebaceous matter protruding from the orifice; similar projections were seen in some of the larger ones on the trunk. The spots could be easily enucleated, and it was perfectly evident that they were in sebaceous glands, but none had fluid sebaceous secretion in any quantity. A few days later many of the spots were larger and more characteristic of the disease. Notwithstanding the copiousness of the eruption, the small size of many of the spots, and the absence of semi-fluid secretion, my conclusion was that the disease was really molluscum contagiosum, and that these minor points of difference were not of real importance. My notes state that he thought he might have caught the molluscum at the Turkish bath.

Case 2.—At the same time (September, 1871) another case of unusually copious molluscum was under care at the Skin Hospital. The patient (Henry T—, æt. 23) had first noticed the eruption under his jaw six months before admission, and a month later it appeared on the legs. When seen by Mr. Tay he had the characteristic spots on the forehead, cheeks, under the jaw, and on the legs; in the last-named situation the spots had ulcerated. He had found out for himself that he could squeeze out the contents of the tumours. There was no history of contagion; he had nothing to do with children, and knew of no one with a similar eruption. He was a compositor.

It would seem not unlikely that in such cases as these the disease may have been contracted at baths, especially the Turkish bath,[1] and in this connection we may notice that all the above patients were men; I have not seen a case of general molluscum contagiosum in a woman.

[1] Mr. Wilson mentions having occasionally seen molluscum contagiosum in men, and says that in one such case the disease was "excited by shampooing."

Case 3.—The subject of this case, a gentleman, æt. 25, was sent to me by my friend Dr. Crosby. He was covered with molluscum spots, which were especially copious on his chest, back, and abdomen. He did not know of any source of contagion.

Case 4.—Mr. S—, æt. 38, had for some months been under the treatment of my friend Dr. —— for syphilis. He had had an indurated chancre, white-edged sores on the tonsils, and a general eruption. Judiciously treated by mercury he got rid of most of his symptoms, but what had appeared to be part of the specific eruption persisted in remaining, and in this condition he was sent to me. I found him covered with a crop of large molluscum spots, many of them somewhat pedunculated. There were none of very small size, all were big enough to excise, and as he was courageous and wished to be well he let me cut away at one sitting upwards of a hundred. More were subsequently removed, and in the course of three weeks he was quite well. He subsequently required further treatment for specific iritis, but he never had any return of the molluscum. This is the only case in which I have ever seen molluscum coincident with a syphilitic eruption, and of course I regard the association as merely accidental. It is, however, quite possible that both were caught at the same time. *December 7th*, 1875.

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2. *Note on the histology of "lepra leprosa" (leprous eruptions).*

By H. Vandyke Carter, M.D.

During a late inquiry into the condition of the skin in leprosy at various epochs of the disease, the following observation was made; it illustrates a very early stage indeed of leprosis.

Case.—A Hindoo from Upper India was admitted into hospital at Bombay for a dysenteric attack, and at the same time a papular or erythematous eruption was noticed on the trunk and limbs, which the man himself hardly regarded, so little inconvenience did it cause. There was feverishness, with irregular exacerbations when the tem-

perature rose to over 100° F. The spots (*vide* Pl. XII, fig. 1) were at first papular, and then formed rounded patches, seldom exceeding an inch in diameter, but having a tendency to bleed; even when small a serpiginous character was noticed in them, and the depressed centre had a dark livid hue; over it the skin was coarsely wrinkled. The spots were very numerous, and they were commonest on the posterior and external surfaces. They came out successively for several days, and the duration of the whole eruption was about fourteen days. For at least two months afterwards slight dark stains in the skin marked the site of the patches, which were then in process of fading; a certain amount of atrophy was now apparent. Desquamation of circular contour had followed, though of insignificant amount. The inguinal glands were tumid for a time. Wasting and weakening of the frame remained; there was no colliquative sweating, &c. The dysentery soon subsided, and the patient has at length resumed the aspect of fair general health. Date, September, 1875.

The man averred that he had never had syphilis, and there were no marks of this specific diathesis about him; he showed no other signs of leprosy. No leprous taint in his family, but there are lepers in his village. It should be borne in mind that no doubt was entertained by both the surgeon in charge and myself of the character of the eruption in this case.

Histology of the exanthem.—I cut out portions of skin enclosing a recent and a faded spot; these pieces were at once placed in alcohol, and next day were examined by means of fine vertical sections, made successively from outside towards the centre.

The recent patch presented the following appearances:—The *epidermal* tissue is everywhere unchanged or but slightly hypertrophied, and its deeper boundary line remains clear and defined. The papillary layer of the *cutis* is at most only flattened out at the seat of greatest intumescence; everywhere vascularity moderate. The most prominent feature of the whole was, of course, abundant cell-proliferation or effusion, and this of a not unusual character; thus, at first it appears around the blood-vessels lying at the base of the papillæ, and here, too, are seen elongated spaces occupied by pale cells; proceeding towards the raised borders of the eruptive patches, the accumulation of cells is found to increase, and finally to occupy the entire thickness of the cutis, together with adjoining parts of the deeper connective tissue; its limits are, however, well defined.

DESCRIPTION OF PLATE XII.

Plate XII illustrates Dr. Vandyke Carter's paper on the Histology of Lepra Leprosa. (Page 297.) From drawings by himself.

FIG. 1.—Spots on the arm, showing the appearance of the eruption at about the sixth day (larger spot); about natural size.

2.—Lymphatics in the deeper parts of the cutis, containing the characteristic "leprous elements." × 500.

3.—View of a section made at the spreading edge of a spot in a vertical direction. Opposite *a* is the unbroken epidermis; at *b* the cutis and subjacent connective tissue; opposite *c* dark brown-tinted particles and masses, scattered in the subcuticular tissues, and seemingly lodged within lymphatic channels; opposite to *d* a group of similar but larger orange-brown masses, which represent the true "leprous elements;" dimensions, $\frac{1}{800}$ in. by $\frac{1}{1600}$ in. They appear to be contained in special cavities, and to be in connection with the smaller coloured collections. × 700. (Treated with liq. potassæ and glycerine.)

Fig. 1

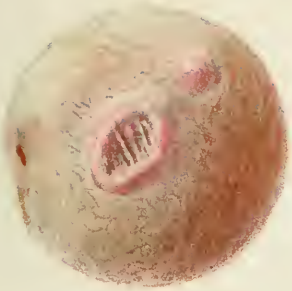


Fig. 2

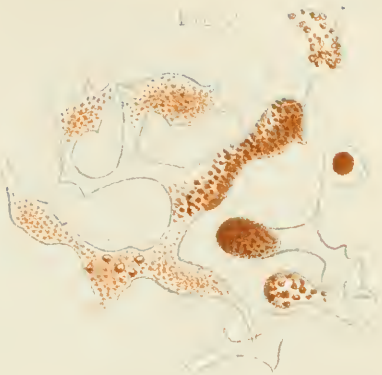
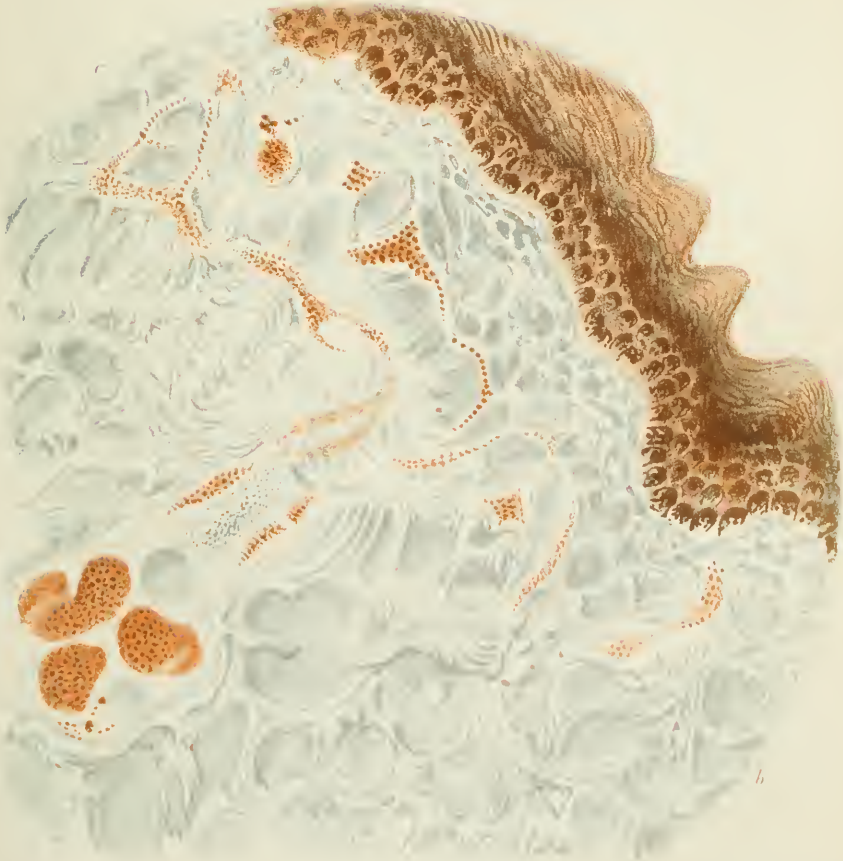


Fig. 3



All appertaining structures seem to be invested with these cells; thus, not only blood-vessels, but sweat-ducts, hair-follicles, sebaceous glands and muscular slips, and also nerve-branches, are seen at the outskirts of the general tumefaction to be coated with layers of cells, which probably occupy their accompanying lymphatic sheaths; traced backwards, these structures, at varying distances, resume their normal aspect. So far as I know, there is here nothing new, nor have I detected amongst these parts a corresponding "irritative" cause which might account for so wide-extended cell-production, comparable, for example, to what has been found in erysipelas, &c. There are, however, foreign organisms which seem to be peculiar; thus, disseminated throughout in tolerable abundance, and best seen at the superficial margins, are minute granular masses of deep orange-brown colour, and arranged in streaks or clusters, evidently, I think, connected with the lymphatic system of channels or vessels. In the same outlying parts they may be sometimes seen in close apposition with the walls of small blood-vessels; they accompany ducts, &c., and many varieties in size and disposition are to be noticed. These coloured bodies may be detected far in advance, so to speak, of the cell-production, and it is in such position that they are displayed in fig. 3; amidst the cellular accumulations they are to be found with difficulty. With the illustrations at hand, a more detailed description seems to be unnecessary. As to the genesis and destiny of these minute structures, I have learnt but little; sometimes it seemed as if they were preceded by delicate micrococcoid material, at other times they appeared to be derived from blood-vessels; and, as regards the later stages of their growth, nothing more than very dark opaque and less diffused and smaller particles have been found in places where the cell-productions have disappeared. This appears on examination of the older and faded patch which I removed.

Acetic acid, solution of potash, and sulphuric ether do not destroy the bodies in question.

Remarks.—Standing alone, the present observation is noteworthy, for I cannot regard these minute organisms as either accidental or strictly homogeneous, at least no bodies like unto them are known to me as occurring in other non-specific cutaneous affections. But the same are found in all the other half dozen varieties of leprous eruption which I have recently examined (mostly in specimens excised during life), and bodies seemingly identical are to be seen in lymphatic

glands removed from the bodies of deceased lepers; they are found too in ordinary leprous nodules ("tubercles") from skin or buccal mucous membrane, or conjunctiva. Hansen has, too, found the like in the retina, liver, and spleen, an observation I have yet to verify for myself in a satisfactory manner; in the testis they are even common. In three or four instances of late I have noticed remarkable changes in nerve branches going to patches of leprous eruption and skin deposit, which, so far as yet examined, seem to be due to the presence of a similar brown-tinted growth; nay, it would appear that the "neuritis" so characteristic of all forms of *leprosis* may be set up by the extension of such growth from the skin along their course, *i. e.* in the outer investment of the nerve funiculi. Such points, and more especially the bearing of these remarks inclusive, upon the pathology of leprosy, must be reserved for another occasion; meantime I may express a conviction that the view of the great leprous disease belonging to the order of morbid infections, which was dimly hinted at by Virchow and has been duly set forth by Hansen of Bergen, is founded upon valid observation, whence necessarily arise inferences of profoundest interest and import.

Bombay, November, 1875.

Postscript to a note on the histology of lepra leprosa by H. Vandyke Carter, M.D.

Since despatching the above note an unexpected opportunity occurred of again scrutinising the condition of the skin in acute leprous eruptions. The patient, a confirmed leper-man, had long been under observation, and quite lately had suffered a prolonged attack of this form of eruption. The subsidence of the skin affection had left numerous livid and depressed (atrophied) marks, which were gradually becoming paler and would probably have ended in *pale* spots, had there not happened in the same places a fresh acute outbreak of the leprous nodules. This relapse was only of short duration, but I took occasion, on the third day after its appearance, to remove from the arm a piece of skin which contained the remains of an old patch and a raised, pinkish nodule, the diameter of a pea, which represented the new outbreak at its onset. This excised portion of skin was at once placed in alcohol, and on the following day thin vertical sections were made, extending from the outskirts to the centre of the spots. After the addition of dilute acetic acid there were seen with a magnifying power of 500 diameters the fol-

lowing pathological appearances in sections belonging to the circumferential part of the leprous growth:—*a*. Copious cell-production of the usual character and position in and below the cutis. *b*. Numerous large, granular, clustered and pigmented masses, of hue passing from orange-brown to almost black, and entirely resembling the "leprous elements" seen in other sections of chronic eruption and of subsiding acute eruptions. *c*. There were streaks of orange-tinted granules, which gave the impression of being newly formed. *d*. Numerous collections of aggregated spherules, in visible characters identical with the well-known *micrococcus masses* or *zooglœa*. These are contained (I quote from my note-book) in defined spaces which in size, form and position, correspond to the lumen of lymphatics and capillaries, bisected at various angles as they pass amidst the tissues; they are numerous and large enough to be regarded as an essential feature. Commonly the vessels or channels are quite filled with these delicate granular masses, but sometimes when partially emptied (by manipulation, as it would seem) the vacant space ensuing is crossed by fine filaments or chains of granules which reveal a tendency, at least, towards a filamentous construction in the masses of zooglœa. Occasionally large vacant spaces, or slits, are noticed, which are evidently dilated lymph-channels, completely emptied of their contents. The direction of such containing channels or spaces is, upon vertical sections of the skin, more or less horizontal, and their position commonly just beneath the true skin, or in it. Transverse sections of the excised portion of skin have not been made, and hence, perhaps, the vessels or channels in question were not often seen to branch or inosculate. A distinct lining membrane, provided with nuclei, was frequently clear enough, and, as well, defined walls either belonging to the vessel (minuter blood-tubes) or formed by condensation (as it seems) of the tissue adjoining the expanded lymph-channel.

A description of the embolic micrococcus-masses is hardly required, since these characteristic growths appeared wholly to resemble the generic form (as it may be called) common to all varieties hitherto made known, and which may probably be regarded as an early stage, afterwards expanding into more species-like forms. In the present instance the growths were often in close association with collections of the dark pigmented bodies, and hence the idea that the new leprous eruption proceeded from relics of the older one.

The epidermis and its appendages were essentially unchanged; sweat-glands and ducts not seen.

I have intimated that these micrococcus-growths are best seen at the edge of the new leprous nodule; such edge is the spreading margin. My mounted preparations are too recently made for transmission to England at present, and on a later occasion I may be able to send them together with drawings in further illustration of the histology of the great leprous disease of man. Further remarks are, therefore, not needed in this place, yet I cannot refrain from observing, in conclusion, that the view of a zymotic origin of leprosy affords a welcome encouragement to our endeavours in checking and preventing a pest which, in India at least, has hardly met with the serious attention which its magnitude and severity clearly call for.

Bombay, November 20th, 1875.

XI.—MISCELLANEOUS SPECIMENS.

1.—*Specimens of organs from a case of infantile syphilis:—interstitial myocarditis, and nephritis; gummata in the liver and lung.*

By Sidney Coupland, M.D.

These specimens were taken from the body of a female child, three months of age, who was brought to the Middlesex Hospital in a moribund condition at 5 a.m. on October 13th, 1875. The infant was at once seen by Mr. Beaumont, then resident obstetric assistant at the hospital. He states that it was then livid, pulseless, with cold extremities, and at its last gasp. It died in a few minutes. Mr. Beaumont has kindly furnished me with the following particulars, which complete the clinical facts of the case. The child was born on the 29th of July, at full term, and had been to all appearances healthy to within three hours of its death, when it began to suffer from shortness of breath. This being unrelieved by a mustard poultice applied by the mother, it was brought round to the hospital. An imperfect account of its having suffered slightly from snuffles, and of having had an eruption on the nates, was elicited from the mother; but it was very plump, and had shown no external signs of syphilis sufficient to lead to its being treated. The child's parents had been married five years; the mother was twenty-six years of age, and the present was the fifth child she had borne. Of these five pregnancies, only one of the offspring, the firstborn, had survived. It was quite healthy. Two were born dead at full term, and there was one abortion preceding the birth of the present infant, which was born at full term, under the auspices of the obstetric department of the Middlesex Hospital. The mother presented no signs of syphilitic taint, and no evidence beyond that afforded by her pregnancies could be obtained as to the existence of syphilis in either parent. The *post-mortem* examination, however, revealed, as these specimens show, an amount of visceral syphilis almost without parallel in so young a subject.

The body was plump and well nourished, the subcutaneous fat

firm and healthy looking. There were traces of a scanty eczematous eruption on the nates and back of thighs. The ribs were slightly beaded. The body weighed 7 lbs. 9½ ozs.

On opening the abdomen the liver was seen to be of large size, even considering the age of the subject. Thus, its right lobe reached to the iliac crest, and the left passed beneath the ribs, reaching the spleen. Visible on the convex surface of the liver, as it lay *in sitû*, was a prominent yellow coloured mass (*vide* Pl. XIII). The organ being removed, this mass, which had a slightly wavy margin and was sharply defined from the hepatic parenchyma, was seen to occupy the greater part of the left lobe and to extend into the right lobe for the distance of an inch, it measuring four inches from right to left and two and a half inches antero-posteriorly. Appearing at the lower border of the left lobe were two nodules, the size of beans, of the same bright orange-yellow colour; and on the inferior surface of the lobe were five other nodules, separated from each other by varying intervals, but proving on section to be outgrowths from the main mass of new material. A similar nodule appeared on the posterior margin of the right lobe. On section this new material was of the same orange-yellow colour throughout, of potato-like consistency and semi-translucent; in no part, not even the centre, showing any opacity or evidence of retrogressive changes. The capsule was nowhere thickened. The remainder of the liver substance was of a dark mahogany-red tint, and was full of blood. The liver weighed 11 ozs. The remaining abdominal viscera were healthy to the naked eye, the kidneys, perhaps, being unduly firm and pale.

The *heart*, weighing 1¾ oz., presented a curious appearance. It was square in shape, apex rounded; both sides contained clot, and the serous layers composing its walls were perfectly smooth and translucent. The myocardium was, however, very firm and resisting, and of a uniform pale pinkish-yellow tinge; the walls of both ventricles and the septum were very thick and cut with a creaking sound.

The *lungs* were non-adherent and perfectly normal in appearance, with the exception of a firm yellow coloured nodule the size of a pea, situated beneath the pleura at the upper margin of the lower lobe of the right lung.

All the organs were submitted to a microscopical examination, of which the results may be thus briefly summed up. The tumour in the liver presented throughout the characters ascribed to young and growing gummata; a fine network of fibrils, enclosing small round

DESCRIPTION OF PLATE XIII.

Plate XIII illustrates Dr. Coupland's case of Infantile Syphilitic Liver. (Page 303.) From by a drawing by Mr. Betts.

The drawing represents the upper surface of the liver, and shows a large gummatous growth involving the greater part of the left, and encroaching upon the right, lobe. Natural size.

cells; a few branched cells, as in myxomatous tissue; and, lastly, some cells in stages intermediate to the small almost nuclear condition to a fully developed spindle or oat-shaped body, were represented in sections taken from different portions of the growth. At the margin of the tumour the adjacent hepatic lobules were invaded by an infiltration of small round cells, passing between the hepatic elements, and occurring massed together, more particularly around the portal canals. The liver cells themselves were many of them vacuolated. In the *lung* the small gummatous nodule presented very similar characters; in it, however, a more fibrous appearance predominated, especially in the central parts. Peripherally, however, small round cells composed the major part of the growth, and invaded the inter-alveolar tissue around it; so that, here and there, the alveoli were reduced to a very small size, in fact almost obliterated, appearing only as irregular spaces left between the rows of cells. At a greater distance from the nodule this interstitial infiltration became less marked, there being a gradual transition from the completely compressed to the normal air cell. Sections taken from the wall of the *heart*, both of right and left ventricle, showed in all parts an extensive infiltration of small round cells imbedded in a structureless matrix, between groups and bundles of muscular fibres. This infiltration was most abundant around the small arteries, but it was by no means limited to their vicinity, for even individual fibres were here and there separated by a row of round cells. The muscular fibres had retained their normal striated appearance, although where the larger tracts of cell-infiltration occurred they tapered off and were lost within it. Essentially the same change obtained in the *kidney*, which to the naked eye had presented a normal appearance. The cortical portion of the gland was the seat of an interstitial infiltration of small round cells; again most abundant around the arteries, and especially around the Malpighian bodies. The renal epithelium was unaltered.

There are many points which make this case one of extreme interest and rarity. In the first place such a wide implication of the viscera is not only rare in infantile syphilis, but is very remarkable as occurring in a child in whom the external manifestations of the disease were so slight, and who had retained its plump nutrition until death. The mode of death, too, is of great interest. Obviously this took place from the impairment of the action of the heart due to the infiltration of its muscular tissue with the syphilitic

new growth. Our 'Transactions' contain but few cases of such a lesion in adults, unassociated with any gummatous tumour in the heart; but in the infant it is infinitely rare. In a contribution to the 'Annales de Dermatologie et Syphilis,' published about two years ago, and summarised by Dr. Pye-Smith in the 'London Medical Record' (1873, p. 764), M. Lancereaux states that in congenital syphilis "nodes have been observed in the substance of the heart, as well as interstitial myocarditis." Gummata in the liver, especially of the enormous size seen in this case, are also very rare in infantile syphilis, the usual affection of this organ being a diffuse interstitial hepatitis,—a syphilitic infiltration similar to that met with in the heart and kidney of this child. *October* 19th, 1875.

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2. *Cystic-oxide calculi* (*cystine*) *removed by lithotomy.*

By Christopher Heath.

The calculi are nineteen in number, three of which were taken from the bladder, and the others from the prostate.

The calculi have all a peculiar waxen look, but the smaller ones are darker and more nodulated than those from the bladder, which latter have a delicate fawn-colour, resembling uric acid, and are distinctly crystalline on the surface. A section of one of the small calculi shows a uniform crystalline surface without any concentric arrangement, but the larger calculi show a deposit of a denser character outside similar crystalline material and arranged in very distinct concentric lamellæ. The calculi of both kinds yielded very beautiful microscopic crystals of cystine, and no other material could be found by chemical analysis (*vide* Report).

The calculi were extracted by median lithotomy, from a man. æt. 28, who was admitted into University College Hospital, June 8th, 1875, with retention of urine. Two years before he was under medical treatment for supposed affection of the kidneys, and passed some blood, and one year before some small stones, not so large as peas, were passed with the water. He had been in good

health until the day before admission, when he got retention of urine, and Dr. Derry Jones, who was called in, passed a catheter and drew off the water, feeling at the time calculous matter in the urethra. Retention recurring he was brought to the hospital, when a catheter again touched a stone. The same afternoon he was cut in the median line by Mr. Heath, who on passing his finger into the wound hooked out of the prostate, partly with the finger and partly with the scoop, sixteen calculi of irregular shape. Passing a sound into the bladder three other calculi were detected and removed with forceps.

A week after the operation the patient had an attack of hæmorrhage which somewhat reduced him, and he made rather a slow recovery, being discharged well on August 11th. The urine was frequently examined during his stay in the hospital; three days after the operation some bright crystals (cystine) were seen floating in it, but on no other occasion was cystine detected.

Calculi of cystine (cystic oxide of Wollaston) are very rare, there being but five specimens in the museum of the College of Surgeons (two of which are only portions of calculi in other museums). Four specimens have been recorded in the 'Pathological Transactions,' all renal, the largest being two shown by Dr. Risdon Bennett, and weighing 204 and 116 grains respectively. They were from an insane patient, and Dr. Handfield Jones, in 1854, showed a specimen at the Harveian Society, also from an insane patient. The calculi are usually small, and occur in members of the same family, for, according to Poland, out of twenty-two collected cases, ten occurred in four families, and in three cases the subjects of the complaint were brothers. Ten specimens in the museum of Guy's Hospital were passed by the same patient, whose age was 30; three were found in the year 1814, six small ones subsequently, and in 1828 another remarkable one of the shape of an ear-drop. Prout gives ten cases, eight between puberty and 47 years, and two in boys.

The largest cystine calculus known is in University College Museum, and was shown to the Society. This when entire weighed over 850 grains, and the half preserved measures $2\frac{1}{5} \times 1\frac{1}{4}$ inches. It was taken from a man æt. 37, by Liston, and was reported on by Dr. Bence Jones in the 'Medico-Chirurgical Transactions' for 1840, where he speaks of "two halves," and remarks that the surface is *not* green. In process of years, from exposure to light, the surface has now become a bluish green, thus resembling the calculus in

Guy's Hospital, which in 1817 was described as brown. Another specimen in University College was from a patient, æt. 19, on whom Liston performed lithotrity. Sir H. Thompson has met with but one case in his practice in a patient æt. 80, on whom he performed lithotrity successfully (specimen shown).

Report on Mr. Heath's specimens of cystine calculi, by Frederick J. Hicks, M.A., Fell. Chem. Soc.—The calculi were nineteen in number and weighed altogether 18·8 grm. (= 290·128 grs.). The largest was of a flattened oval shape and measured 51·7 mm. by 42·2 mm. in its greatest diameter. The weight of the two halves together was 8·409 grm. (= 129·85 grs.). This is referred to as (*b*) in the subsequent examination.

The next in size was in section, square-shaped, and its diagonals measured 18·3 mm. by 17 mm. The two halves together weighed 2·55 grm. (= 39·382 grs.). This was marked (*a*).

There were four others of nearly the same size, and the remaining thirteen varied from the size of a horsebean to that of a large shot.

At a glance they could be separated into two groups, one apparently consisting of calculi found in the prostate. The surfaces of these were irregular and nodular, and one tapered off into a process about 8 mm. long. The other group contained only the three larger calculi, which from their smooth rounded appearance may be presumed to have been some time in the bladder.

The following are the weights of the principal calculi:

Two halves of calculus (*a*)	2·55	grammes	=	39·352 grs.
„ „ (*b*)	8·409	„	=	125·85 „
„ „ (*d*)	0·64	„	=	9·876 „
Calculus (*c*) undivided	0·74	„	=	11·111 „
„ (*e*) „	1·74	„	=	26·851 „
Smallest calculus .	0·025	„	=	0·3857 „

In those which had been divided allowance must be made for loss in sawing, polishing, testing, &c.

On looking at their structure as they appear to the naked eye externally and on section there are seen to be two distinct forms of deposit; one is composed of large translucent crystals of a fawn-brown colour, the other also is evidently crystalline, but the crystals glisten only on the exterior surface, especially

when wetted or when viewed by candle-light. This deposit is stone-coloured, and is much closer in texture than the other. The second deposit only occurs in any quantity in the larger calculi, and in them the exterior is more regular and even. The denser form is deposited in connective layers, of which in the section of calculus (*a*) as many as fifteen or twenty can be counted, while in the large calculus (*b*) the layers are thicker and not more than five or six in number. The smaller calculi have irregular nodular surfaces, and do not present the same glistening appearance, although here and there their crystalline structure is very evident. On section, these appear to be homogeneous and also more compact than the inner portion of the large calculus, but some of the smaller ones are slightly encrusted over with the denser form of deposit.

It is worth noting that in the larger calculi the extra size is not merely due to a thick deposit of this denser layer, but the central portion itself is in them absolutely larger than the smaller or prostatic calculi which are composed of but one form of deposit.

The following tests were applied to determine their chemical character:—Portions of the external layer and of the central portion were scraped off from the large calculus marked (*b*). The samples of powder so obtained when examined under the microscope appeared alike, but presented nothing characteristic. Both samples dissolved entirely in dilute ammonium hydrate (liq. ammoniæ), leaving no residue at all. Phosphates and cholesterine were thus proved to be absent. The solutions so obtained appeared faintly opalescent, but no apparent cause for this could be observed, nor did microscopic examination reveal anything. To both solutions hydrochloric acid was added in excess, and they were then covered and set aside for twenty-four hours, but no precipitation occurred of anything that could be detected either by the naked eye or under the microscope.

Samples from the two layers of calculus (*a*), of the outer layer of (*c*), and some of the dust obtained by sawing through (*d*) were also treated in precisely the same manner. In each case there was the same faint opalescent appearance and the same negative result as regards the finding of either cholesterine phosphates or uric acid.

Portions of various parts of different calculi were then incinerated on platinum foil, but no residue was left. Another and much larger sample of powder obtained by sawing through (*d*) was burned on platinum foil. In this case a very small amount of residue was left,

This was examined under the microscope, but it did not appear to have any definite structure: still as it was the pulverised calculus that was burned and not a chip of the substance, this result is not altogether conclusive. It was far too minute in quantity for any examination to be made as to its chemical nature. On first applying heat to the sample an evolution of gas took place from the mass, so that if care was not taken the whole was blown away. This took fire and burned with a purplish flame, a residue of porous carbon being left which rapidly burned off. The platinum for a moment was stained of a purplish-black colour, but this was very transient, and on completing the combustion the surface of the platinum was unaltered. At first an evident smell of hydrocyanic acid was observed, but this was rapidly succeeded by a most offensive smell, the nearest resemblance to which is that obtained during sawing or filing vulcanite.

The specific gravities of the large calculus marked (*b*) and of the smaller one marked (*d*) were taken.

Sp. gr. of large vesical calculus (*b*) = 1·649.
„ prostatic calculus (*d*) = 1·734.

From these facts it appears, therefore, that both deposits consist of pure cystine, and this result is confirmed by the specific gravities which were obtained, the mean of the two numbers (1·6915) corresponding very closely with the sp. gr. of a pure cystine calculus examined by Venables. The sp. gr. of this calculus is given in 'Watts's Chem. Dic.' as 1·7143. The method which yielded the best specimen of cystine crystals under the microscope was to dissolve the powdered substance in a dilute warm solution of potassium hydrate (Liq. Potassæ), and just neutralise the still hot solution with acetic acid. On cooling brilliant crystals of cystine were deposited. These were washed several times by decantation with cold water, and a drop of water containing the suspended crystals was allowed to evaporate on a slide. The crystals were mounted dry and in various media. Of these dammar varnish and glycerine were found to be the best. The sharpness of the forms of the crystals seemed to be lost when a saturated solution of calcium chloride was used, and when mounted dry the crystals were generally too opaque to be examined with a high power, as is necessary with the small crystals which are obtained by precipitation with acetic acid from the solution in ammonium hydrate.

All the specimens were examined with a Hartnack's microscope, using ocular No. 2 and objectives Nos. 4 and 8.

Nov. 2nd, 1875.

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3. *Syphilitic gummata in liver, spleen, and kidneys.*

By W. S. Greenfield, M.D.

The organs, which were exhibited as fresh specimens, consisted of the liver, spleen, and kidneys removed from the body of a female, 25 years of age, who died in St. Thomas's Hospital from the effects of thrombosis in the cerebral arteries. They afforded an example of the early growth of numerous gummata in the kidney; a somewhat rare situation.

The notes of the case were necessarily incomplete, as the patient died forty-eight hours after admission to the hospital.

Aves A—, a married woman, æt. 25, was admitted on December 2nd, 1875, under the care of Mr. Simon, for advanced syphilitic necrosis of the nasal and superior maxillary bones. She walked into the ward, and nothing was noticed to give rise to a suspicion of cerebral disease. Shortly after admission, whilst in bed, she was seized with a "fit," became unconscious, and speedily lapsed into a state of coma. Remedial measures were employed in vain, and the patient died on December 4th, at 3.25 p.m. Next to nothing could be ascertained as to her previous history, except that she was separated from her husband, and had suffered from disease of the nose and palate for four years.

Post-mortem.—December 6th. Body somewhat emaciated. No trace of old or recent eruption on any part of the body, and no scars on legs or elsewhere. No nodes on tibiæ or clavicles. Vulva free from sores, glands in groins not enlarged. Mucous membrane of vagina smooth, with almost entire loss of rugæ; no mark of ulceration. Uterus and ovaries normal.

Septum nasi almost completely destroyed; nasal bones and cartilages necrosed; an irregular fissure half an inch in width, extending through the hard palate for 1½ inches backwards from the alveolar

border of the upper jawbone, the latter being also partially necrosed and the incisor teeth loosened.

Tonsils and pharynx with some scars of old ulceration.

The condition of the thoracic organs presented nothing noteworthy.

The *liver* was of normal size, weighing 46 ozs.; and of dark colour. The capsule for the most part normal, free from thickening; but on the upper surface of the right lobe were several small slightly depressed and puckered cicatricial patches, much less depressed than usual with syphilitic scars. At the posterior border of the right lobe was a small yellow nodule the size of a pea, encapsuled by fibrous tissue. On cutting into the organ several gummatous nodules were found in the centre of the right lobe. These were rounded, firm, smooth and of yellowish-white colour on section (closely resembling the section of a horse-chestnut), limited by a delicate zone of fibrous tissue, which was less than $\frac{1}{2}$ a millimètre in thickness in the largest. The largest of these nodules was 16 millimètres in diameter, two or three others were about 10 millimètres, and some smaller, from 2 to 5 millimètres. One of them was adjacent to and had partially infiltrated the wall of a large branch of the portal vein, causing a slight prominence on its inner wall, but not penetrating it completely. The remainder of the hepatic tissue was of normal appearance and did not stain with iodine.

Spleen.—Considerably enlarged, weighing $7\frac{1}{2}$ ozs., somewhat pale, capsule slightly thickened. At its upper part was a large infarct occupying nearly one quarter of the whole of the organ, extending across it, slightly prominent on the surface. On section, this infarct was found to be partially decolorized, though comparatively recent; the arterial branch leading to it was completely blocked by a firm whitish-yellow clot.

On section of the remainder of the gland the following appearances were observed:—For the most part the healthy tissue remaining was of rather pale colour, with distinct Malpighian corpuscles; but the greater part of the organ was in one or other of the following conditions. Small yellow translucent patches rather sharply outlined, rounded or irregular in shape, varying from 3 to 6 millimètres in diameter, and having all the characters of gummata in an early stage. Other patches of larger size, but varying much in size and shape, many situated near the surface of the organ, and somewhat ill-defined; the surface of their section of dead-white

colour, or with a yellowish or purplish tint, and somewhat granular. These latter patches in some cases were evidently connected with obstructed vessels, the walls of which were greatly thickened. It was impossible to say from naked-eye examination how much was due to new growth and how much to obstruction of the vascular supply.

Kidneys.—Both a good deal enlarged, but of normal shape, together weighing 15 ozs.; capsule slightly adherent, surface irregular. On section they presented a very curious appearance. Throughout the cortical substance were very numerous patches of pale yellow beeswax-like colour, many of them rather sharply outlined and surrounded by a zone of congestion. The smaller patches were for the most part in narrow lines radiating outwards from the bases of the pyramids, whilst the larger ones were of very irregular shape and occupied a more or less wedge-shaped tract of the cortical, and in some cases of the medullary, substance. In some of them the outlines of vessels with greatly thickened walls could be traced, and the smaller tracts also appeared to follow the direction of arterial branches.

In addition to these there were also numerous patches of dead white or slightly reddish colour and granular appearance surrounded by a zone of intensely congested tissue, resembling, in fact, the condition which would be produced by comparatively recent arterial obstruction.

The *aorta* was somewhat atheromatous at its commencement, the patches of thickening being rounded, elevated in the centre, but not very sharply outlined. On cutting through them they appeared thicker than usual, and presented only very slight degeneration of their deeper layers. Throughout the remainder of the aorta was general slight atheroma of ordinary character.

The vessels at the base of the brain were for the most part free from ordinary atheroma; but the left middle cerebral artery from near its origin, and some of its main branches were converted into firm white fibrous cords, the lumen being greatly narrowed, and occupied by a somewhat recent thrombus. The thickening of the walls of the vessels had little of the appearance of ordinary atheroma; it was white, and extended for some distance along the wall, involving the whole circumference of the vessel, the walls of which were considerably thickened, here and there only a very narrow lumen remaining. There was no trace of fatty degeneration or calcification, the vessels were not especially adherent to the pia mater. The corresponding

tract of brain was very extensively softened, but the precise limits need not be detailed.

A rough examination of the new growth in the various organs showed that it had the characters of syphilitic infiltration, but the author expressed his intention of bringing the results of a more careful microscopic examination before the notice of the Society on a future occasion. *December 7th*, 1875.

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4. *Case of arrested development of the bones of both fore-arms; extreme senile changes in the osseous tissue.*

By Alban Doran.

These bones are from a subject dissected in the spring of this year at the Royal College of Surgeons, for the Court of Examiners.

It was the body of a female pauper, æt. 54, who died on the 27th of April, 1875, at St. George's Workhouse, Kensington.

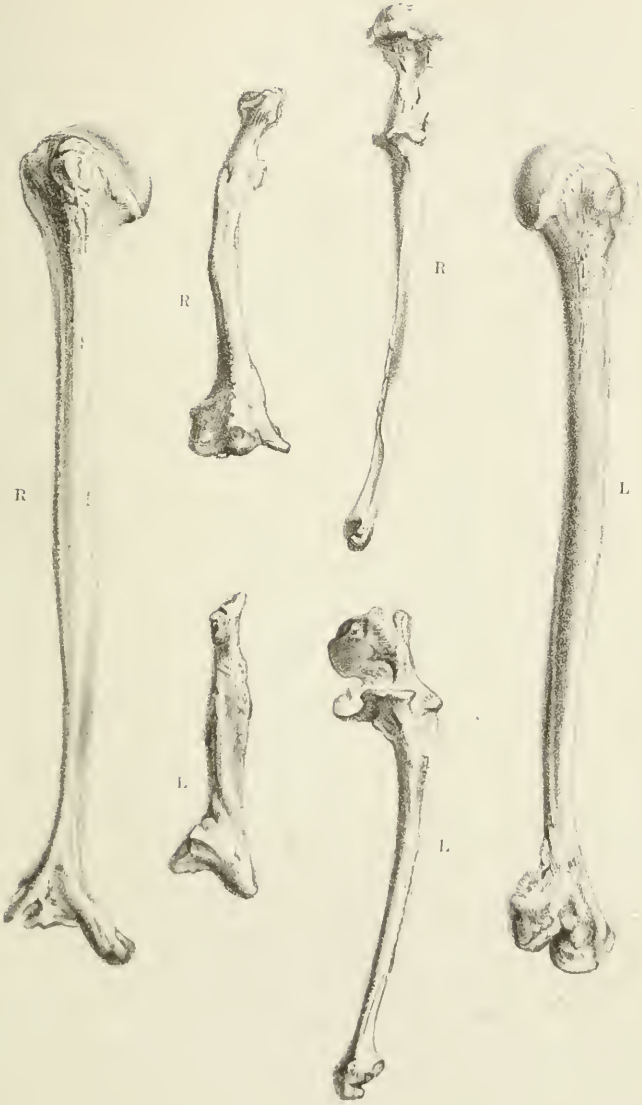
On applying to Dr. Dudfield, of Kensington, who attended the patient before she died of pulmonary disease, he kindly informed me that she had told him that her arms had always been deformed.

When the subject was brought up to the theatre of the college I was struck by the singular deformity of both upper extremities. On extending them and drawing them downwards, the middle finger on the right side could only just be made to touch the great trochanter, and on the left side it could not reach so far.

There was not a trace of any scar on any part of the integuments on either side, and all the articulations were freely moveable. The muscles of the forearm were rather pale though not ill developed, and arose from their normal origins; the vessels and nerves were all normal in their course. Both hands were large and presented no deformity. On opening the elbow, the radio-ulnar and radio-carpal joints, the synovial membrane and cartilages appeared normal, but the bone beneath was very soft and could be readily broken down between the finger and thumb.

DESCRIPTION OF PLATE XIV.

Plate XIV illustrates Mr. Alban Doran's case of Arrested Development of the Bones of both Forearms. (Page 314.) From drawings by Mr. Sherwin.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On maceration a great quantity of the osseous tissue in the neighbourhood of the articulations was washed away, including the whole of the head and neck of the left radius.

The humeri are a little diminished in length in proportion to the rest of the skeleton; they measure about 9½ inches each, the body being 5 feet 2 inches in height. They show no signs of rickets, nor could I detect any trace of that disease in any other part of the body (*vide* Pl. XIV).

The lower ends of these bones have been much dissolved in maceration, particularly the right side. What appear like inflammatory deposits on the left are portions of bone harder than the rest, which have resisted disintegration.

Both radii are very short and distorted, the right measures not quite 4½ inches, which is exactly half the average length as given by Professor Humphrey. The left has suffered in maceration even more than the humeri. In both, the carpal ends are enlarged and distorted.

The ulna is more undeveloped on the left side than on the right. The shaft of the right ulna is tolerably straight, its articular surface for the humerus is very wide and the olecranon unusually long, the margins of the articulation are shallow. The styloid process is very short and bent inwards.

The left ulna has a shorter and more crooked shaft than its fellow; its muscular attachments are well marked.

The upper end is very large in proportion to the rest of the bone, the olecranon is broad, and the articular surface for the humerus is very deep. The articular surfaces for the radius are very shallow on both sides[1] (*vide* Pl. XIV).

There seems to be no doubt that the changes in the bone tissue of these specimens are senile; the patient was 54 years old and prematurely aged—a condition where such signs of decay are often particularly well marked.

It might be suggested that the deformity may have originated in infantile paralysis or in rickets.

But in the first complaint there would be atrophy of the muscles of the forearm and hand, and the bones would be slender and devoid of rough surfaces for muscular attachments. Here all these conditions are reversed.

[1] The entire specimen may now be seen in the Museum of the Royal College of Surgeons of England, Pathological Series, No. 941 B.

There were no traces of rickets in any other part of the body, the skull, spine, and pelvis were normal, and the tibiæ, so generally involved in that disease, were straight and slender. I have never heard of a case of rickets completely localised to one pair of extremities.

Evidence seems to me in favour of the shortness being due to arrest of development. The patient owned that her arms were in this condition all her lifetime; possibly the arrest was fœtal and temporary, so that after birth the bones continued to grow, maintaining at the same time their defective proportions to the whole skeleton.

It would, in that case, resemble the fœtus exhibited before the Pathological Society by Mr. William Adams, in the Session 1872-3, but there the legs were involved. Hence it would come under the class commonly known as "Nanomelus," or, as Mr. Lowne more simply expresses it, "arrest of growth affecting the limbs," but here, contrary to what he states to be most usual, there is some defect in development as well, for the disproportion between the upper and lower halves of both radii is not entirely explained by the extreme senile changes in the former.

December 21*st*, 1875.

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5. *Trachea, showing absence of thyroid gland, and fatty tumours, from a case of sporadic cretinism.*

By Fletcher Beach, M.B.

About two years ago I had the pleasure of bringing before the Society certain organs taken from a sporadic cretin, among which was a trachea showing absence of a thyroid gland. In that case the swellings above the clavicles which usually are present in this disease, and which had at one period of the child's life been evident, disappeared before death and were not found *post-mortem*. I have now the pleasure of bringing before the Society another case of sporadic cretinism and exhibit the trachea, showing absence of a thyroid gland, and two fatty tumours which were found in the posterior triangles of the neck. It is the fifth *post-mortem* of the kind on record, and the results bear out the observations made by Mr. Curling, Dr. Langdon Down, and Dr. Hilton Fagge. The photographs exhibited were taken after death (*vide* Woodcut 8).

E. R. S—, a girl, æt. 11, was admitted into the Clapton Idiot Asylum, May 26, 1875, and died November 18th, 1875, of effusion into both pleuræ, and pneumonia of a low form of the left lung. The following history was obtained from the mother:

"The child was born on September 30th, 1864, being the third of four children by the first husband, the two elder children being boys, the youngest a girl. They were healthy when born and showed no sign of cretinism. They died of various diseases. There is one child by the second husband alive, and to all appearance healthy. This child was born in London and has never been out of it. She had seven fits when a baby, ascribed by the mother to teething. Since then she has been healthy. She cut no teeth till she was 2 years old. According to the mother's account she has always been well fed. She did not grow from 15 months to 5 years old; then she grew up to 8 years of age.

No history of phthisis, neuroses, or mental disease on either the father or mother's side. They were not related before marriage. The father, who is dead, was very intemperate and is said to have become more so before this child was born. He was intemperate at times of connexion. He had "a running" several times, but there appears to be no history of syphilis."

The mother is an exceedingly robust woman, above the average height, and according to her own account has always been healthy.

No instruments were used when this child was born.

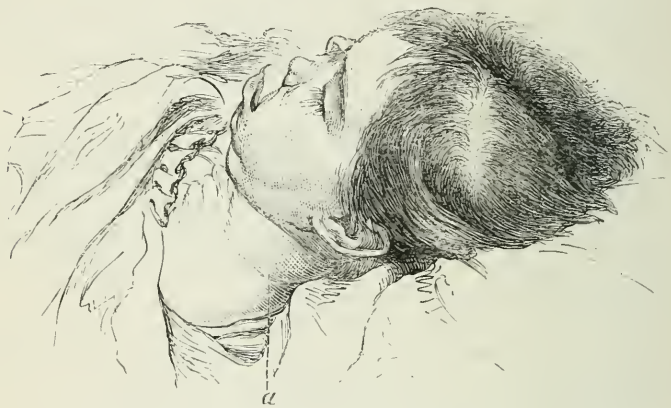
The following was the child's condition:

She was 32¾ inches in height and weighed without clothes 2 stone 4 lbs. She was very well nourished, having a considerable amount of fat in the abdominal walls. The head was large, flattened at the top, spreading out at the sides. It was covered with sparse, auburn-coloured hair. It measured 9 inches in the longitudinal diameter, 10⅜ inches transversely between the tips of the ears, and 19⅝ inches in circumference.

The anterior fontanelle was not quite closed, there being a depression about the size of a sixpence in that position. The sutures were not well united, a depression being felt in front of the anterior fontanelle in the middle line. A prominence could also be felt along the line of union of the coronal suture. The forehead measured 2½ inches in height and 5¼ inches in breadth. The distance between the tip of the forehead and the chin was 6 inches. The countenance was vacant and void of expression. Complexion

sallow, except at times, when her cheeks became flushed. Eyes blue, with long eyelashes. Nose exceedingly pug-shaped: scarcely any bridge could be felt. Ears large, but not larger than normal for a child of her age. Lips thick, generally slightly apart. Gums very spongy, but the teeth regular, small in size, and in fairly good condition. Tongue of normal size. Arch of palate flattened. Cheeks full and flabby; a layer of fat beneath the chin, forming a "double chin." Not the slightest trace of goitre, but there were two well-developed swellings, one on each side of the neck, above the clavicle (*vide* Woodcut 8, *a*).

Woodcut 8.



a. Shows one of the fatty tumours as it appeared during life.

The arms and legs were short and curved. The former measured $9\frac{1}{2}$ inches from the point of the shoulder to the wrist. The latter $13\frac{1}{4}$ inches from anterior inferior iliac spine to external malleolus. The skin in these positions was thick, easily separable from the muscles, and of a deep red colour. The general colour of the body was sallow. The hands and feet were small, the palms of the hands measuring $3\frac{3}{4}$ inches and the soles of the feet $4\frac{1}{2}$ inches. The labia were well developed. There was no sign of rickets.

The abdomen was distended, measuring 21 inches at the umbilicus. The child had no control over either motions or urine, which were passed under her.

In the daytime she usually sat up in a chair, requiring no support, and towards the last took some notice of what was going on in the ward. She was placid and quiet. She could not speak, but if dis-

turbed would make an articulate sound, which, however, bore no resemblance to any word. She could not stand nor walk. She slept well.

On removing the scalp at the autopsy the anterior fontanelle was found to be not quite closed, the inner extremities of the coronal suture being also membranous at this part, so that there was a lozenge-shaped piece of membrane of this shape and size. The remainder of the coronal suture was evident by a line of redness. The sagittal suture was quite evident behind the anterior fontanelle, while in front of it in the middle line was a piece of membrane about $\frac{1}{4}$ inch in width, the frontal bones being not yet united. The centres of ossification of the parietal bones were well marked by vessels diverging from a centre.

On removing the skull cap it was found to be thicker anteriorly and posteriorly than laterally, being $\frac{1}{4}$ inch in thickness in the former positions, and $\frac{1}{8}$ inch in the latter. It was flattened at the top and correspondingly on its inner aspect, it was somewhat bulged inwards. The sutures on this aspect were marked out by depressions. The skull cap was adherent to the dura mater along the line of the frontal suture and at the anterior fontanelle, as well as for a space of $\frac{1}{2}$ inch on each side of the middle line, so that it was removed with some difficulty. On examining the base of the cranium, the opening of the foramen magnum was seen to be smaller than normal, but its margin was surrounded by an elevated rim which gave to it a triangular shape. The rim was especially prominent at its front and back part, there being in those positions two prominences with a groove between.

The space enclosed measured, together with the rim, $1\frac{1}{2}$ inch laterally, and $1\frac{1}{4}$ inch antero-posteriorly. The basilar process, $\frac{7}{8}$ inch in length from before backwards, was nearly horizontal; it was narrowed laterally, and the anterior and posterior clinoid processes were on the same level.

The sella turcica was only $\frac{1}{2}$ an inch in length from before backwards. The anterior fossæ were normal, but the middle fossæ were prominent in the centre. The cerebellar fossæ were flattened. The base of the skull, it will be seen, was therefore considerably altered in appearance. On measuring its length internally it was found that $2\frac{1}{2}$ inches represented the length from the anterior border to the anterior margin of the sella turcica, and 4 inches the length from the posterior part of the sella turcica to the posterior border

of the skull; 7 inches, therefore, represented the entire length, including the length of the sella turcica.

The brain was firm and weighed 31 ounces, the cerebellum, pons, and medulla contributing $4\frac{1}{2}$ ounces of this weight. The convolutions were exceedingly coarse, measuring from $\frac{3}{8}$ to $\frac{1}{2}$ inch in width, the measurement being taken in their course, not at the point of bending. They, as well as the sulci, were well marked The posterior lobes of the brain overlapped the cerebellum to a considerable extent. There was no congestion of vessels and no excess of fluid in the ventricles. The pons and medulla were smaller than usual, corresponding with the small size of the basilar process and foramen magnum.

A portion of the brain was submitted for microscopical examination to my friend, Dr. Savage, Assistant Physician to Bethlem Hospital, and subjoined is his report.

Cerebral Convolution of Cretin.—Pia mater thickened and adherent. Vessels tortuous. Cortical layer thicker than usual.

Pyramidal cortical corpuscles normal, with rather large nuclei.

The corpuscles in the more superficial layers are surrounded by larger spaces than usual.

No general wasting and no signs of inflammatory change.

GEO. H. SAVAGE.

The trachea was of small size, and showed not the slightest sign of a thyroid gland. Its place was taken by a little fat. In each posterior triangle was a fatty tumour, differing from that surrounding it, being pinker in colour (*vide* Woodcut 9). The tumours, though lobulated, were not encapsuled, and hence there was some difficulty in defining their limits. They both sent processes backwards and upwards beneath the sterno-mastoid muscles, as well as downwards beneath the clavicles. They did not extend into the axillæ.

The heart, lungs, liver, spleen and kidneys were healthy.

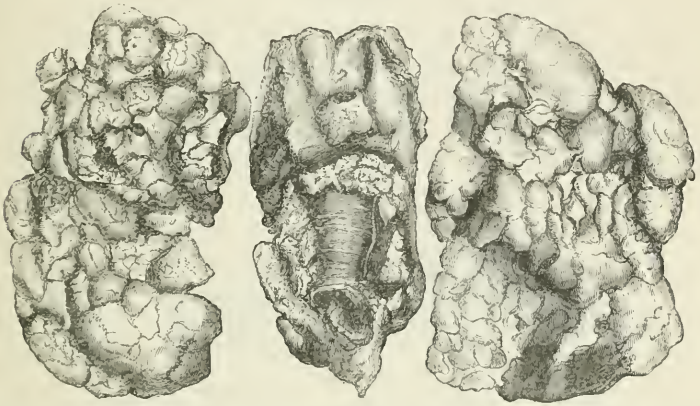
The ovaries were larger than normal for a child of her age. That on the right side contained a cyst.

It will be seen from this description that the horizontal position of the basilar process, the dwarfing of the sella turcica and the elevated rim round the foramen magnum closely correspond with the description given by Dr. Hilton Fagge of the base of the skull of a sporadic cretin, whose case he brought before this Society nearly

two years ago. These characteristics also closely correspond with those found in endemic cretinism.

Sporadic cretinism, though to some extent rare, is not so rare as might be expected. I have at present two cretins in the Clapton Idiot Asylum, and I have notes of three or four whom I saw when Medical Registrar at the Hospital for Sick Children, Great Ormond

Woodcut 9.



Street, and only a short time ago, by the kindness of Dr. Barlow, Assistant Physician to that hospital, I had the opportunity of seeing another case of the kind.

The intelligence, though limited, in these cretins varies considerably in amount. In the case which I have related it was very small, but one of the cretins in the Clapton Asylum answers questions quite rationally and goes to school in the asylum.

The causation of sporadic cretinism is still somewhat obscure. Various theories have been given to account for its existence, but my impression is that many cases will require to be collected and studied before a reliable explanation can be given.

January 4th, 1876.

6. *Artificial teeth removed from the larynx and passed by the bowels.*

By Christopher Heath.

The two gold tooth-plates exhibited disappeared from the mouth of a young lady, æt. 21, during an epileptiform attack in the night (*vide* Woodcut 10). They consisted of the two right incisors mounted on a gold plate and fastened by a pivot to the central tooth (fig. 2), and of the left central incisor and canine teeth also mounted upon a gold plate fastened partly by a pivot and partly by a gold band (fig. 1). The latter plate was swallowed and passed through the

Woodcut 10.

Fig. 1.

Fig. 2.

bowels without difficulty on the fifth day. The plate shown in fig. 2 passed into the larynx and became impacted in the cricoid cartilage, giving rise, however, to such slight distress that its presence was only recognised definitely with the laryngoscope. It was removed by tracheotomy on the fourth day and the patient made a complete recovery.—See Clinical Lecture by Mr. Heath, 'Medical Examiner,' January 28th, 1876. *January 18th*, 1876.

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7. *Lardaceous reaction in the dysmenorrhœal membrane.*

By John Williams, M.D.

The specimens exhibited are dysmenorrhœal membranes. One, the thinner and smaller of the two, was taken from the uterus of a young girl who had died of pleurisy. It was given me by Dr. Sydney Coupland. The patient died on the fifth day of menstruation, and

the uterus contained besides sanguineous débris two pieces of membrane. The one exhibited was the larger of the two and was partially adherent to the wall of the uterus. It presents the characters of the so-called mucous membrane of the uterus. It is the kind of dysmenorrhœal membrane most commonly met with.

The other specimen is a rarer variety—the apoplectic form. It is a complete cast of the uterine cavity. On cutting into it a very fine canal is detected in its interior running from the part which corresponds to the inner orifice towards the part corresponding to the fundus. In that situation the canal divides, each tube corresponding to a horn of the uterus. The structure of the decidual membrane in this specimen is not so apparent as in the first described because the whole tissue is infiltrated with blood; still the decidual structure may be discovered on careful examination. I have been induced to show these specimens because it has been asserted recently by Dr. Finkler, of Kiew, that the pathological condition in dysmenorrhœa membranacea is lardaceous degeneration, and the evidence of this is that the membrane gives a blue reaction with sulphuric acid and iodine. When I first examined the membranes with a view to decide this point, I found I obtained the blue reaction constantly along the side of the specimen and everywhere on the glass slide (for I examined microscopically) where the iodine and the acid came in contact. The membrane itself was not coloured in a characteristic manner except at the edges. I further found that menstrual blood gave the same colour with the reagents, and finally, that the reagents themselves even gave it in perfection. Virchow was aware of this reaction between iodine and sulphuric acid, and endeavoured to distinguish between it and the lardaceous reaction by the granular appearance of the colour produced by the reagent alone, while the colour is uniform when lardaceous matter is present. It is true that in the coloured field produced by iodine and sulphuric acid alone granules appear, but the fluid around the granules is also coloured; and it is difficult if not impossible to distinguish between the colour produced by the reagents alone and that produced by the reagents with lardaceous matter.

I have brought these specimens before the Society, and have shown the colour produced by the action of sulphuric acid on iodine alone, in order to show the worthlessness of this colour test of the presence of lardaceous matter. *May 18th*, 1876.

8. *Specimens of lardaceous organs presenting some unusual characters, with observations on the causes of the lardaceous change in general.*

By C. Hilton Fagge, M.D.

Case 1.—*Lardaceous liver, from a case of old spinal disease with abscess. Atrophy, and fatty degeneration and calcification of parts of the organ, all reaching its surface.*

On April 22, 1875, I made an autopsy in the case of a young man, æt. 24, who died of dropsy after curvature of the spine. He had had spinal disease when four years old, and ever since there had been a discharge of pus from a sinus in the right groin.

The diseased vertebræ proved to be about seven in number, from the sixth dorsal to the second lumbar, all of which were anchylosed together, so as to produce a rounded prominence in the back and a sharp angle in front. The mass thus formed had an irregular sinuous cavity in its midst, lined by a smooth reddish membrane, and containing numerous loose pieces of bone or calcareous grit. An abscess extended down in the sheath of the right psoas muscle.

The abdomen was greatly enlarged, contrasting remarkably with the small shallow chest. The liver weighed 163 ounces. The anterior part of its upper surface was flattened by the depressed state of the lower ribs. Its tissue was highly lardaceous. Most of it was of a red colour, but some parts were white or yellowish, and opaque. These were all on the surface of the organ; its anterior edge was so affected, and particularly the lobulus spigelii. The opaque white parts generally faded off gradually into continuity with the rest of the liver, but in some places they were rather sharply defined. They were generally much depressed below the normal level, and thus they really looked not unlike syphilitic patches. But on section it was clear that they consisted entirely of lardaceous hepatic substance, which had undergone a fatty change. Several of these whitish-yellow parts contained a large quantity of calcareous matter, in the form of irregular granules, which were gritty to the touch and against which the knife grated.

The kidneys and intestine were lardaceous, but not the spleen.

Under the microscope I found that in the yellow or white parts of the liver the lardaceous glistening character of the cells was well

marked, but that they also contained numbers of minute opaque granules. The calcified portions appeared structureless; they looked crystalline, but without definite form. The addition of dilute nitric acid caused effervescence.

CASE 2.—*Lardaceous spleen, presenting wedge-shaped whitish-yellow patches, like ordinary infarctus.*

On March 1, 1876, I examined the body of a man, æt. 32, who died in the hospital of Bright's disease. He had been admitted with general dropsy. His urine was in quantity about four pints daily, of sp. gr. 1015; it contained so much albumen that, when coagulated, this occupied about half the bulk of the urine. The swelling had subsided soon after his admission, but he was attacked with diarrhœa and sickness and he gradually sank. I found that there were numerous small softening nodes beneath the pericranium. The liver contained from eight to twelve whitish-yellow tough gummata, with soft mucoid centres, of no great size, but of the most characteristic appearance. The proof of syphilis was thus complete. The testes were healthy, and no cicatrix could be detected on the genital organs.

Several of the viscera were lardaceous. The liver yielded a slight and partial reaction with iodine. The right supra-renal capsule was affected with the change in a marked degree, the left was not noticed.

The spleen, which weighed $10\frac{1}{2}$ ounces, was highly lardaceous, presenting the appearance known as the "sago spleen." It also showed some six or seven whitish-yellow spots, or patches, of very irregular forms and sizes, but all reaching the surface. Their appearance contrasted sharply with that of the rest of the organ: and my first impression was that they might be gummata. However, on section they were found to be more or less wedge-shaped, like infarctus. They gave a reaction with iodine to about the same extent as other parts of the spleen. On section their cut surface was of a dull yellow colour, with some greyish translucent parts. It therefore appeared clear that they were simply portions of the lardaceous organ which differed from the rest in being in a state of fatty degeneration. The microscope, however, showed that the degenerated structures were not those which were already lardaceous, for these still retained their glassy appearance. The parts which looked opaque and granular were those which lay between the sago-

grain spots. It is to be added, however, that the difference between the whitish-yellow patches and the rest of the spleen was much less marked under the microscope than it appeared to the naked eye.

Case 3.—*Lardaceous liver, some superficial parts of which formed defined waxlike nodules, undergoing atrophy, and separated from the rest by deep grooves so as to be mistaken for gummata.*

On June 1st, 1875, I made an autopsy in the case of a man, æt. 44, who had been admitted into the hospital for heart disease, and who was found after death to have the aortic valves adherent together and nodulated with large masses of calcareous deposit. This affection was probably of rheumatic origin, as he had had acute rheumatism four years previously. There was no history of syphilis, although he had confessed to having had gonorrhœa and a bubo sixteen years before. Both testes, however, were fibroid, and the left one contained several gummata. The spleen, intestine, and kidneys were lardaceous. The liver weighed 105 oz.; it was firmly adherent by old connective tissue to all the parts around it. It did not present the ordinary appearance of a lardaceous organ, being of an orange-brown colour and devoid of translucency, and looking homogeneous on section, without evident lobular markings. However, iodine gave an abundant reaction with all parts of it, and the microscope showed that a large part of its secreting tissue had undergone the lardaceous change. On its surface, and also in its interior, it presented several whitish nodules which I took at the time for gummata. Those which lay imbedded in the substance of the organ were comparatively small and few in number; they appeared to me to have been of very recent development, being surrounded by purplish-black zones of intensely congested hepatic tissue. None of these happened to be present in that part of the organ which was preserved for more minute examination, and which showed a number of the superficial nodules. This is much to be regretted, for when the latter were carefully inspected it became at once evident that they were not gummata at all, but simply portions of the lardaceous hepatic tissue, which had undergone further changes. Some of them were rounded, others irregular, in outline. They were from $\frac{1}{2}$ to $1\frac{1}{2}$ inch in diameter. One of them penetrated for $\frac{1}{2}$ an inch into the substance of the organ; others less deeply. This one was convex on the surface; its centre only reached the level of the rest of the organ, so that its margin was necessarily below

that level, and was separated from it by a deep groove. The nodules themselves were whitish-yellow and had a smooth waxlike appearance. Their substance generally appeared to be continuous with the hepatic tissue; but towards the surface of the organ, where there was a groove, the two parts seemed to be separated by a tolerably definite line of connective tissue.

Under the microscope it was at once apparent that the whitish-yellow nodules consisted simply of hepatic tissue which had universally undergone lardaceous change, or from which all unchanged liver cells had disappeared by absorption.

There was no trace of fatty degeneration.

Case 4.—*Lardaceous liver, having on its surface a raised white patch, containing large calcareous masses, and looking as though it occupied the thickened capsule of the organ, but really consisting of altered hepatic tissue.*

The Museum of Guy's Hospital contains a preparation of a lardaceous liver presenting some peculiar characters, which led to its being exhibited by Dr. Wilks at one of the meetings of the Pathological Society.[1] His description of it is that "the right lobe, which was adherent to the diaphragm, showed a firm white deposit on the surface, resembling suet, and that on this was some cretaceous deposit. It was two thirds of an inch in thickness, and it was spread on the surface of the liver to the extent of a space as large as the hand. On examination it was found to be pure lardaceous material and exhibited no hepatic structure, but was composed of rounded masses of the adventitious substance. It could not be said with certainty whether the material being in excess had destroyed the hepatic structure in which it had been deposited, or whether it had been an altogether free formation beneath the capsule of the liver."

Dr. Wilks was lately kind enough to draw my attention to this preparation, and I have subjected it to a fresh examination. It presents obvious points of resemblance to two of the specimens already described; but there are also points of difference which at first sight seem to prevent one's placing it with them.

The substance of the liver generally is affected with the lardaceous change in a moderate degree; but part of its surface is white and has an altogether different appearance from the rest.

On section this peculiar appearance is found to affect a layer half

[1] 'Path. Trans.,' xv, p. 133.

an inch in thickness; which, however, thins off gradually at its edges. It is not perfectly homogeneous, but presents here and there opaque fibrous striæ. Imbedded in it are several calcareous nodules of irregular form, as large as peas or beans. Parts of these are exposed upon the surface of the organ, where they appear as opaque yellow smooth bodies, convex and slightly raised above the level of the rest.

So far there is little to distinguish this specimen from the others. But on the exterior of the liver the area presenting this peculiar appearance has at its edge a projecting lip, which strongly suggests the idea that it is superimposed upon the original surface of the organ. And this conclusion is strengthened by the fact that at least on one side it is separated from the hepatic substance by a definite plane of connective tissue, which looks like a prolongation of the capsule of the liver beneath it. And under the microscope one finds that even when this boundary seems less distinct there are fibrous bands which intersect the masses of liver cells, for a small depth, very much as in cirrhosis. This fibrous tissue does not itself react with iodine, but the vessels contained in it turn of a deep mahogany brown colour. On the other hand, the white substance superficial to it is universally affected by iodine. It consists of a glistening material of an indefinite structure, apparently consisting of a network of fibres which have undergone complete lardaceous transformation.

Still it is to be remembered that in Case 3 there was also a line of connective tissue which probably separated the white nodules from the rest of the liver; and I have come to the conclusion that in both instances it is to be regarded not as any part of the capsule, but as a new formation, or as resulting from the condensation of masses of the interlobular connective tissue, after absorption of the corresponding columns of secreting cells. There seems to be no doubt whatever that the white substance itself consists of extremely lardaceous hepatic substance.

Case 5.—*Mixed lardaceous and cirrhotic liver, part of which was so altered as to simulate the appearance of syphilitic gummous nodules.*

Another preparation in the Museum of Guy's Hospital was placed there by Dr. Moxon. He took it out, in 1869, from the body of a young woman, æt. 28, who died of chronic affection of the liver and kidneys, the result of disease, apparently syphilitic, which had come on since her marriage six years before. The liver, spleen,

intestine, supra-renal capsules, and kidneys were all lardaceous. The liver weighed 70 oz.; it was greatly altered in shape, bound down by a very thick capsule, and closely adherent to the diaphragm. On removal of the capsule deep seams and scars showed themselves; and when these were cut into, many of them were found to lead down to yellowish and cheesy patches which were at first regarded as gummata. In many parts there was an appearance of opaque cheesy substance in the lobules themselves, as if from a general syphilitic hepatitis.

Under the microscope, however, Dr. Moxon found that the apparent gummata was really only portions of the hepatic tissue, which had undergone the lardaceous change in an extreme degree. He made beautiful drawings of the appearances, both to the naked eye and with $\frac{2}{3}$ inch objective, and these he kindly lent me for exhibition to the Society. Some of the lobules in the opaque white-yellow parts were entirely lardaceous; others had some of their cells unaltered. There was a great increase of fibrous tissue, with some small-cell-growth between the lobules, exactly as in ordinary cirrhosis.

These cases show that lardaceous organs are liable to undergo certain further changes, affecting only parts of their substance, the nature of which changes may easily be misunderstood. In Cases 3 and 5 it was at first supposed that gummata was present; in Case 2 the altered tracts in the spleen might easily have been mistaken for common infarctus; and in Case 4 the affected portion of the liver was regarded as an independent deposit of pure lardaceous material upon the surface, or in the capsule, of the organ.

It appears to me to be pretty certain that these peculiar appearances are in part due to want of arterial supply. That such is the case is evident from the form of the patches in the spleen in Case 2, and from the fact that in almost all of the other cases the affected parts were all on the surface of the liver, where they could receive blood only on one side. But it would seem that another element in the production of the waxlike patches in the liver is the occurrence of the lardaceous change in the tissue of these parts in an extreme degree,—and indeed universally,—though the rest of the organ may be affected with it to a moderate extent only. Why this should take place only towards the surface of the liver I do not comprehend.

With regard to the nature of the further changes in the whitish-

yellow patches, it has not seemed to me that fatty degeneration plays any necessary part in their production. In Case 4 the lardaceous tissue appeared to be undergoing calcification directly, and without the occurrence of any intermediate degenerative process.

Indeed, although many English pathologists have been inclined to assume that lardaceous viscera are specially liable to undergo fatty degeneration, I am not aware on what evidence this opinion rests. Rindfleisch says that in the liver "amyloid and fatty degeneration are not seldom found associated:" but he adds,—"the latter usually presenting the characters of an occasional and accidental complication." Uhle and Wagner indeed mention that "the lardaceous degeneration of the blood-vessels in glandular organs often leads to secondary disturbances in the glandular epithelium (simple atrophy and fatty metamorphosis)." But they go on to say that the results of defective blood supply are but little noticeable:—except in the intestines, where peculiar "lardaceous ulcers" arise; and perhaps, very rarely, in the liver.

Case 6.—I have, therefore, thought it worth while to make a fresh examination of a preparation in the Museum of Guy's Hospital, which is described in the catalogue as a specimen of a liver "partly fatty, partly lardaceous." Dr. Wilks placed it on the shelves, and his account of it is that on the surface it was pale, looking like a fatty organ; but that on section its interior contained large masses of lardaceous matter. The spleen was affected with commencing lardaceous change. The patient, a man æt. 25, had died in the hospital of the effects of chronic disease of the hip-joint with abscess and fistula.

On examination of this specimen I find that (as Dr. Wilks states) the opaque parts, which have the appearance of an ordinary fatty liver, are chiefly towards the surface; while those which look simply lardaceous are towards the interior. There is no definite boundary line between them: they intersect one another freely, and there are many parts which are mottled irregularly with both appearances. Under the microscope no part of the organ is found free from lardaceous change: even those portions which look most opaque have small foci of lardaceous degeneration scattered through them. Still the amount of this is very small in comparison with that which is present in the more transparent portions of the liver, for in them only the portal channels and the very centres of the lobules retain their

natural structure ; and all the hepatic cells which remain are small and shrunken. On the other hand, the opaque parts are mainly made up of aggregations of hepatic cells loaded with large oil drops, exactly as in an ordinary fatty liver.

It seems to me difficult to give a satisfactory explanation of these appearances. They do not seem consistent with a retrograde change in the liver tissue, consecutive to arrest of blood supply; nor do they indicate a fatty degeneration of structures which had before become lardaceous; and on the other hand we can hardly continue to regard them as the result of an accidental conjunction of the lardaceous and fatty affections of the liver; for it is difficult to understand why the former change should be less considerable in these parts which happen to be affected with the latter.

Hitherto I have not observed in lardaceous kidneys appearances similar to those above described in the liver and the spleen. But I am nevertheless inclined to think that these observations may throw some light on a point which has long been a puzzle to me, namely, the great frequency with which the morbid processes that belong to other forms of Bright's disease are found associated with the lardaceous change in these organs. In the liver and in the spleen, when partially affected by this condition, the rest of the parenchyma is commonly quite healthy ; but in the kidney, I think, it scarcely ever happens that interstitial and epithelial nephritis are absent when the arteries and Malpighian tufts are lardaceous. Are these changes the result of disturbance of the blood supply to the organ? In my second case, which clinically had passed for an instance of the ordinary chronic mixed form of Bright's disease, the kidneys weighed $12\frac{1}{2}$ oz.; they were very uneven on the surface; their tissue was mottled with opaque whitish yellow. They gave a reaction with iodine, but the degree of lardaceous change did not at first appear to be considerable. I thought that they were specimens in which such a change in slight amount was associated with an advanced general epithelial nephritis. But under the microscope all the Malpighian tufts were found to be highly lardaceous; many of them were converted into shining globes; the arteries supplying them were affected for a considerable length, and even the larger trunks. Some of them were distinctly obstructed by an accumulation of granules and even of fatty globules in their interior. The opaque yellow appearance was due to the presence of a large num-

ber of corpuscles of Gluge, which were scattered in the renal substance. Many of the Malpighian corpuscles were surrounded by thick layers of fatty granules. After hardening with chromic acid these appearances were less obvious, but some of the Malpighian bodies were then seen to have a growth of small cells extending for some distance round them, just as in the early stage of ordinary interstitial nephritis. The epithelium was opaque and granular, but its nuclei could still be seen. Many of the tubes themselves were highly lardaceous.

The remaining preparation is one which has no direct bearing on the questions discussed in this communication; but as it is affected with the lardaceous change, and I believe it to be unique of its kind, I take the opportunity of exhibiting it to the Society with the other specimens.

CASE 7.—*Lardaceous fibroma of a splenculus* (?)

The specimen is an oval rounded smooth tumour, which weighed about 36 oz., and was taken from the body of a man, æt. 41, who died in Guy's Hospital under the care of Dr. Moxon in November, 1874. The presence of this tumour had been the cause of his admission into the ward. It was moveable and occupied the left hypochondrium. The man himself was much wasted, with sunken cheeks and eyes. The blood was found to contain an excess of white corpuscles. He had diarrhœa and vomiting. His urine was of spec. gravity 1010, and contained much albumen.

At the *post-mortem* examination it was at once apparent that the tumour was neither in the spleen nor in the left kidney. The former weighed $3\frac{1}{2}$ oz.; it presented scattered lardaceous Malpighian bodies. The kidneys probably were slightly lardaceous, and so were the liver and small intestine. The large intestine showed numerous scars of former ulcers of dysentery, from which he had previously suffered while in India. The protracted suppuration attendant upon this disease was doubtless the cause of the lardaceous change in the viscera.

At this stage of the examination, therefore, there seemed at first to be no alternative but to suppose that the large tumour must be an enlargement of lumbar glands. It was whitish yellow on section, firm and dry, yielding no juice. It seemed to be made up of two distinct portions, separated by a slightly sinuous line. But

the application of iodine brought out numerous mahogany brown points which were just like Malpighian bodies of the spleen; and Dr. Goodhart, therefore, suggested that the tumour might perhaps be due to morbid change in an accessory spleen, or rather perhaps in two "splenculi," which, becoming enlarged, had got flattened against one another; and it seems to me that, strange as such an affection is, it is scarcely possible to take any other view.

Microscopical examination showed that the tumour consisted mainly of fibrous tissue, without any definite arrangement. The lardaceous parts had the usual glassy appearance.

Dr. Wilks very kindly made the suggestion to me that this tumour might be similar to one that existed in the mesentery in a case which he brought under the notice of the Society in 1864, and which forms Case 4 in the present series. But I have re-examined that specimen, and find that the growth is a lymphoma, and that its tissues are entirely free from lardaceous changes.

In the discussion on syphilis I made some remarks based upon the assumption that lardaceous changes in the viscera are found only in two sets of cases, the one consisting of persons who have been affected with syphilis, the other consisting of those who have been the subjects of prolonged suppuration. I have since found that the members of the Society were not prepared to occupy this position without reserve; and that writers on pathology, although they lay stress on the frequent association of lardaceous changes with the two causes which I have mentioned, nevertheless admit that they may depend upon other conditions. Thus Uhle and Wagner mention, as very rarely giving rise to these changes, leuchæmia, enteric fever, rachitis, obstinate malarial fever, organic heart diseases; and they add that such changes occur independently of any primary lesion in Bright's disease.

My remarks in the debate on syphilis were almost unpremeditated, and I was then speaking solely from my own personal experience in the *post-mortem* room of Guy's Hospital during the last five years. But it has since seemed to me that the question was worthy of fuller investigation, and I have, therefore, asked my friend Mr. Lancaster to be so good as to search the records of the autopsies made at the hospital for a longer period. In the meantime Dr. Dickinson has brought forward at the Royal College of Physicians some statistical facts with regard to the causation of lardaceous

diseases, derived from observations made at St. George's Hospital. But his cases are less numerous than those which Mr. Lancaster has collected; and I do not find it stated in what proportion of them the change was not traced to either of its recognised causes.

The results which Mr. Lancaster has evolved for me from the *post-mortem* records of Guy's Hospital are as follows:

During the twenty-one years from 1855 to 1875 inclusive there have been 244 cases in which lardaceous changes in the viscera were present, besides two cases in which the evidences of such change were slight or doubtful, so that they may be omitted from consideration.

Of these there were 154 in which chronic suppuration had existed for a considerable length of time, but in which there was no evidence of syphilis. This number is made up of

Cases of Phthisis	67
Joint disease (generally either hip- or knee-joint)	29
Disease of vertebræ (in most of them psoas abscess existed)	12
Caries or necrosis of other bones	10
Pelvic abscess (cellulitis)	6
Empyema, discharging externally	3
Scrofulous disease of kidney	4
Dysentery	3
Tubercular ulceration of intestine	3
Bedsores, following fracture of spine	2
Abscess of liver discharging externally	1
Suppurating ovarian cyst	1
Carbuncle of eight months' duration	1
Empyema and pneumo-thorax, scrofulous kidney	1
Compound fracture of leg (3½ months.)	1
Old running abscesses and fistula	1
Stricture and old cystitis	1
Chronic pyæmia and abscess in areolar tissue	1
Ulcerating cancer of uterus	1
Ulcerating sarcoma of abdominal wall	1
Ulceration of leg	1
Iliac abscess (cæcal in origin)	1
Dilatation of bronchi and suppuration	1
Discharging sore in neck, caseous disease of glands	1
Calculous pyelitis	1
	154

In addition there were 5 in which some suppuration had been present, but in which it was doubtful whether this had been sufficiently profuse and of long standing enough to cause lardaceous changes: namely,

Case of stricture with suppurating kidneys . . . 1
(In this the history is very imperfect; it is not stated whether there had been continued cystitis or not.)

Case of abscess of liver, not discharging externally . . 1
(The man had been in India, but there was no history as to whether he had had dysentery.)

Case of tubercular peritonitis and chronic caseous disease of mesenteric glands 1

Case of inflammation and abscess of testis from blow; of two and a half months' duration, but only opened two weeks before death 1
(The history of this case is very imperfect.)

Case of chronic deafness, with discharge from one ear; and of "stoppage of nose," which had lasted for some time, and had been attended with discharge 1
(This is the case which Dr. Wilks brought before the Society in 1864, and it is Case 4 of my series of fatty and calcified lardaceous organs. There was a tumour of the mesentery, which is described as "fibro-cellular." I have re-examined this, and find that the peripheral soft part of it is a typical lymphoma, while the central hard part is made up principally of fibrous tissue.)

5

(There were about 6 of these cases in which the origin of the suppuration was some local disease, having a definite starting-point, so as to allow of one's forming an opinion as to the length of time required for the development of the lardaceous change in the viscera. One patient had had a carbuncle eight months, another had suffered from pelvic cellulitis for exactly the same period. A third had had a bedsore seven months, the result of a fracture of the spine received a fortnight previously. In a fourth case there was an ulcerating sarcoma of the abdominal wall, which had been discharging for four months only. In a fifth there had been fracture of the spine three months before, bedsore for two and a half months, and a double empyema: but this patient had fibroid testes, and a scar in the groin. Lastly, a sixth case was one in which amputation of the leg had been performed three and a half months before death, on account of a compound fracture, with abscess; in that instance the patient died of diphtheria, and it is particularly noted that the lardaceous change was just commencing in both the liver and the spleen.)

On the other hand, there were during the same period 76 cases of lardaceous diseases of the viscera in which there was satisfactory proof (either from other *post-mortem* appearances, or history, or both) of the existence of syphilis.

Mr. Lancaster noted for me that in about 34 of these there was evidence of former or present bone-disease or suppuration, leaving about 42 in which the affection seemed attributable to the syphilis *per se.* (In the twenty-one years there were altogether 177 *post-mortem* examinations in which syphilis seemed to have been present; so that the proportion of cases of syphilis in which lardaceous disease existed was nearly 43½ per cent.)

The evidence of syphilis in these cases was, of course, of varying degrees of completeness. There were 3 other instances in which there was at any rate a suspicion of its presence.

Case of gout with large red kidneys and fibroid testes . .	1
Case of cirrhosis of liver (patient had had a chancre six years before, and was said not to have been intemperate) . . .	1
Case of cachexia and "kidney with enlargement" of ulna; with apparent gummata in spleen	1
	3

The cases in which there was evidence either of syphilis or of suppuration, therefore, amount altogether to 238, and there remain only 6 in which no such evidence was made out.

In one of these, however, in which no other disease seems to have been found except the lardaceous changes in the liver, kidney, and spleen, it is notable that the testes seem not to have been examined; and the same may be said also of another instance, in which cirrhosis of the liver was present. And it may be that cirrhosis is itself an occasional result of syphilis, for that affection existed in a third case, the patient being a woman æt. 28. In a fourth case, again, there was a small ulcer of the stomach; but as it measured only an inch by a quarter of an inch, one could hardly suppose that it had secreted a sufficient quantity of pus to cause lardaceous disease of the viscera. In that instance, however, it is noted that the last two true ribs on one side were considerably enlarged, as well as their cartilages; this was perhaps due to syphilitic periostitis, and it is to be noted that in this case neither the skull nor the testes were examined. In each of the remaining two cases death occurred from acute mischief, altogether unconnected

with the lardaceous disease; in the one of pyæmia after amputation of the thigh for a diffused popliteal aneurysm, in the other of acute pneumonia and pericarditis.

Considering how very variable in different cases is the amount of evidence by which the presence of syphilis is established in the *post-mortem* room, we surely cannot be surprised if lardaceous changes in the viscera should sometimes be the only indication of it, and if the changes in question should nevertheless be of syphilitic origin. To give such an explanation of the few instances that cannot be otherwise accounted for, out of a total number of 244 cases seems to me far more reasonable than to refer them to some as yet unknown cause; and we should then have a very important addition to our means of identifying syphilis in the dead subject.

On the other hand, it is noticeable that, with scarcely one exception, fibroid disease of the heart and aneurism of the aorta, which some believe to be effects of syphilis, are wanting in the series of cases which Mr. Lancaster has collected. This surely tends to show that the supposed relation between these changes and the disease in question has no real existence. *May 2nd*, 1876.

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9. *A specimen of tænia mediocănellata.*

By F. Charlewood Turner, M.D.

The specimen exhibited was brought by an out-patient under treatment at St. Thomas's Hospital.

An examination of it showed that, while the general appearance of the entozoon was quite normal, one portion of the broader part was remarkable by the absence of any indication of segmentation.

At the top of the preparation is a segment showing the normal character; below this are two segments imperfectly separated by a notch on one side only; below this, again, there is a length of about 14½ inches, in which a similar imperfect indication of segmentation appears at the upper and lower portions only, both borders of the animal being unbroken for a distance of 12 inches between. Below this a third portion of the animal is shown, in which seven

segments appear to have been similarly united in a length of something less than 3 inches.

It is also noticeable that the sexual apertures are arranged here with the greatest irregularity. Thus, out of seventeen orifices counted in the larger portion, at one part six are crowded together in the small space of little more than $1\frac{1}{2}$ inches, while lower down they are absent for a distance of $4\frac{1}{2}$ inches, and on one border none appear for over 10 inches. A typical development of the sexual apparatus corresponding to only one of them can be seen.

May 16*th*, 1876.

XII.—SPECIMENS FROM THE LOWER ANIMALS.

1. *Fatty tumour from the pectoral muscle of a hen.*

By Edwards Crisp, M.D.

I removed this tumour from the pectoral muscle of a living hen; it had existed for a long time, but of late had increased rather rapidly. It weighs 4½ oz., and is composed entirely of large lobules of fat.

As far as I know, it is the first example described of a fatty tumour from one of the lower animals. *Nov. 16th*, 1875.

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2. *Rickets in young pheasants.*

By Edwards Crisp, M.D.

I have examined recently several young pheasants, the bones of which I exhibit. These birds were hatched under hens, and had a limited space, there being too many on the same ground; they, moreover, had not the ants' eggs and other insects they would have obtained if they had had a larger range. The long bones of the lower extremities near to the joints were large and spongy, and so deficient were they in ossific material that two birds about six weeks old that were brought to me alive had their legs broken from the inability to sustain the weight of the body. The spinal bones and bones of the upper extremities were but slightly affected. Although many of these birds died, a large number of them recovered, the distorted bones ultimately becoming solid and strong.

As is generally the case in children that labour under rickets, these birds suffered from unnatural food and defective assimilation, and hence the deficiency of solid matter in the bones.

I remember a curious example at the Zoological Gardens, Regent's Park, of a young ostrich that "fell to pieces," to use the keeper's expression: the ribs were so soft and deficient in phosphate of lime that the chest collapsed and the bird died very quickly.

Nov. 16th, 1875.

3. *Fractured humerus of a gorilla.*

By Edwards Crisp, M.D.

This fracture is in the middle of the humerus of a gorilla. The bone is united with very little deformity. The fracture was oblique, and about 4½ inches in extent, the upper end being within 3½ inches from the head of the bone, and the lower end 6 inches from the olecranon. This example of a well-united fracture in a wild animal is one out of many that I have seen in quadrupeds and birds, where the union has apparently been as perfect and the limb as useful as if splints had been used. I also place on the table specimens of ulceration and fracture of the phalanges of the upper extremity in the chimpanzee and ourang, these injuries being very common in the anthropoid apes from their propensity to fight with their teeth. On looking over many skeletons and bones I have found several of these injuries.

Although not pathological, I place likewise on the table for the inspection of those who may be interested in the matter the largest *humerus* of the gorilla I believe ever seen. It weighs 18½ oz., and its length is 17½ inches. On comparing this with several human *humeri* I find that the arm bone of a man about 6 feet in height weighs 5 or 6 oz., and its length is about 13 inches. *Nov. 16th*, 1875.

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4. *Larynx and trachea from a dog dying of measles.*

By William Squire, M.D.

Four cases of croup under my notice during the past month, with its sudden changes of temperature, show the influence of season in determining the incidence of this disease as sequel to whooping-cough and measles. The only case to recover was one of great severity after measles; relief occurred when false membrane was coughed up, but there was further danger from capillary bronchitis, and after that from meningitis. The very day I pronounced the child out of danger from these sequels of measles died the pet dog that had been in the room and licked the hands of this child while the rash was fully out. The dog's symptoms, commencing twelve days after contact with the child, and the *post-mortem* appearances of the specimen exhibited—the intense congestion, in rounded spots, of the bronchial, tracheal, and laryngeal mucous surfaces, without false membrane, so closely correspond with the results of measles, that the facts may be worth putting on record. *May 2nd*, 1876.

XIII.—DISCUSSION

ON

THE PATHOLOGY OF SYPHILIS.

February 1st, 1876.

Mr. Jonathan Hutchinson opened a Discussion on the Pathology of Syphilis. He said:—In engaging in a discussion on syphilis, with a view to the advance of our knowledge of it, there are certain facts as to its nature which we may, I presume, take as generally accepted. It is, I suppose, now a matter of general belief that the phenomena of this disease, notwithstanding their great variety in detail of character, are due to one virus, which, having been introduced into the body by contagion, developes within it,—with tolerable uniformity as regards stage and time,—certain processes of inflammatory growth. The pluralistic doctrine of Carmichael never obtained any wide acceptance; and it is now, I think, generally acknowledged that what was called dualism had not really any claim to such a name, since but few ever believed that there were really two rival forms of virus capable of producing constitutional effects. Further, even in its much more restricted sense, as implying that there are two distinct kinds of virus, to which are attributed two different forms of chancre, I think we may say of dualism that it is dead, and that the far simpler creed which attributes the soft chancre to contagion with inflammatory products produced by syphilis, but not, as a rule, containing its germs, is the one which now obtains general acceptance. We have, then, in syphilis, but one malady and one virus.

The objections which, on account of its apparent irregularity, were at first urged against the claim of syphilis to take position amongst the specific fevers, have been greatly diminished of late

by facts from two different directions: first, by the observation that the short-lived exanthemata are by no means so regular and uniform in their career as it had been customary to account them; and that, in point of fact, they do vary quite as much as to length of stage, degree of severity, and occasional omission of some of their phenomena, as does syphilis. Secondly, we have come to appreciate more correctly the antidotal power of our specifics, and have remembered that it is not fair to allege against a malady that it is irregular in its course, when our observations are made on cases in which we have done our best, by means of a most potent antidote, to arrest its career. I have no doubt that, if mercury were entirely put aside, we should soon see that syphilis is quite as regular in its stages as variola, and also that it varies quite as little in its degree of severity in different persons. When mercury does not cure, it delays; and the retardation of stages sometimes witnessed under its use is very remarkable.

Thus, then, what I shall have to say this evening will be based on the assumption that in syphilis we have to deal with a specific fever of prolonged but definite stages, which is produced by contagion only, which has a period of incubation, a period of outbreak (known as primary symptoms), and a period of efflorescence or exanthem (known as the secondary stage); and which, in exceptional cases, differs somewhat from its more short-lived congeners by being followed by sequelæ to which we give the name of tertiary symptoms.

Had it not been for this unfortunate tendency to recur and become protracted, syphilis might have remained a surgical malady. If it always ended, as it ought to do, with its exanthem or secondary stage, there would have been no need to trouble physicians as to its treatment, nor is it very probable that the Pathological Society of London would have thought it worth while to invite a detailed discussion of its life-history problems. To the annoyance and misery of thousands, however, syphilis does not always end with its apparent death at the expiration of the secondary stage. The patient seems then to be well; he loses his fever; his rash disappears; his throat heals; his hair grows again; and his blood is renovated; but he is not safe. There remains ever after a risk—comparatively a very small one, but still sufficient to cause much anxiety—that he may become troubled with a tendency to the growth of tumours, which will be in direct relation to his bygone

disease. These may occur in important parts, may attain large size, and will probably show very little tendency to spontaneous cure. To these we give the name of tertiary symptoms or sequelæ; and we recognise a most important distinction between them and those of the earlier stage, in that they are never general, and only by accident symmetrical. They do not constitute another stage of a blood disease; but, by their constant non-symmetry, appear to prove that now, at least, the blood is not concerned. We no more witness returns of the syphilitic exanthem—a symmetrical and general eruption—at long periods after the first, than we do recurrences of the smallpox eruption after the patient has recovered. Most unhesitatingly may we characterise as errors in observation a certain small number of cases which have been supposed to illustrate this occurrence. In all probability syphilis ceases to be a blood disease at the date when symmetrical manifestations cease to be usual. The precise period at which this takes place will vary with the treatment pursued and the idiosyncrasy of the patient.

The correct determination of the period at which syphilis ceases to be a blood disease could, perhaps, be accomplished with certainty only by inoculation experiments. That there is a period at which it does so cease without in the least exempting the patient from the risk of fresh local symptoms, appears highly improbable. Whilst the secretions from all forms of secondary ulcerations and the blood of patients in the secondary stage are abundantly proved to be contagious, the negative evidence as regards both in the tertiary stage is very strong. The secretion from a phagedænic sore in a tertiary case may spread phagedæna, but will not spread syphilis; and hence, probably, one frequent source of what are called soft sores. It would be rash to assert that it is impossible for tertiary syphilis to prove contagious, but at present I know no evidence which would support the affirmative. The test of contagion is probably the best that we possess as regards the existence of a blood taint, and guided by it there seem strong reasons for suspecting that risk of hereditary transmission may persist long after the cessation of blood contamination. At any rate, the risk of contagion appears to cease long before the risk of hereditary transmission. I admit that there is difficulty in conceiving of this, or in understanding how a taint no longer active in the parent's fluids can be reproduced with virulence in his offspring, but the facts seem to point in that direction. In speaking of the possibility of local

syphilitic phenomena occurring long after the cessation of blood-taint, it is intended to imply very definitely the belief that many of the late phenomena of syphilis are purely local; and that, if they exist in parts which are accessible to local remedies, they do not imply any necessity for internal treatment. If I may be pardoned a statement which refers to clinical rather than to pathological matters, I may say that the long persistent and frequently relapsing sores on the tongue, and many forms of skin diseases, palmar psoriasis, lupoid tubercles, &c., are local and not constitutional, and are cured quite as easily by local as by internal treatment. The internal gummata are also probably often only local, but you cannot bring mercury or iodine into contact with them, excepting by introducing these remedies into the blood. It is scarcely necessary to remark that success by internal treatment by no means proves that the disease was more than local.

Before proceeding further to speculate upon the true relationship between the secondary phenomena and their sequelæ, it may be best to glance briefly at some of the peculiarities of the syphilitic inflammations. So peculiar are the products that one is almost tempted to speak of new growth rather than of inflammation. From beginning to end—from the chancre to the latest tertiary gumma—the tendency to cell-growth is most remarkable; and the production of a solid palpable mass, often very firm, is a characteristic feature of the inflammatory process when lighted up by the virus of syphilis. It is, however, by no means the only one. An avoidance of proclivity to suppuration (not constant, but still very marked), a tendency to cause death of the tissues affected, and thus produce phagedænic ulceration, or even sloughing, and a proneness to undergo rapid and complete absorption, especially when attacked by certain metals or their salts, are features which characterise and distinguish the new growths due to syphilis. Let us add that, whilst absorption may easily be procured, relapses are very prone to occur, especially if the treatment be arrested too soon. It is to be insisted that these peculiarities are the common property of syphilis in all its stages. A gumma in the tongue may be almost as hard as a chancre, and either of them may be, and have been many a time, mistaken for scirrhus. A chancre, when very large and dense, may perish and slough out just like an overgrown gumma. The chancre, the secondary rash, and the gumma are all alike liable to undergo molecular death and to be attended by

phagedænic ulceration. To attempt to distinguish between the secondary and tertiary stages, by saying that the latter only is attended by liability to deep ulceration, is to set facts at defiance.

The attempt to found distinctions as to the stage of syphilis upon the tendency or otherwise to deep ulceration, has been the means of introducing much confusion into the subject. It cannot be too strongly insisted, that this may be shown at any stage. It may be remarked also, in passing, that phagedænic action constitutes an exception to the statement that the inflammations due to syphilis in an early stage show a tendency to spontaneous cure. When the wizard Syphilis has once called up the demon Phagedæna, it has evoked that which it is powerless to control. No law of spontaneous cessation and recovery will now be observed; nor will the specifics for the parent malady avail much as regards the offspring. Phagedæna, once started, exists on its own behalf, and spreads by the contagion of its own pus. To arrest its spread it is necessary to destroy its secretion.

So marked is this liability to phagedæna in syphilis, and so rare in connection with any other cause, that with a few exceptions we may count syphilis, either directly or indirectly, as the parent of all phagedæna. The discussion as to whether rupia and its allies are secondary or tertiary symptoms may thus be easily decided, by explaining that there is nothing in the "rupial sore" characteristic of either, and that its features may be assumed in both. If rupia occur as a symmetrical eruption, it will always be found that the period which has elapsed since the contagion is only short, and that the disease is consequently secondary; but if it be unsymmetrical, then in all probability it is tertiary. Histologists, I believe, admit that the differences in cell-structure between an indurated chancre, a secondary tubercle, and a tertiary gumma, are only very slight, and that essentially all three are constructed on the same plan.

Thus, in examining the question as to the relationship between the several stages of syphilis, it is, I think, a matter of necessity that we admit that many features and many tendencies are shared by the phenomena which occur in all. Whilst, however, their similarities are marked, so also to some extent are their differences. First, and as by very far the most important of the differences between the stages, we place one which has been already mentioned, the tendency to general and symmetrical development in the secondary stage, and to local, restricted, and unsymmetrical formations in

that of sequelæ. This, however, after all is merely a question of abundance and not so much of character. In the secondary stage the blood and all the tissues are involved; whilst, in the later ones, only certain regions, or it may be only single spots, are affected. The second difference is, that a spontaneous tendency to resolution of the new growths and to absorption is constantly witnessed; whereas it is exceptional in all tertiary products. It is, however, by no means certain that spontaneous disappearance does not often occur in the case of tertiary growths. Still it may be fairly granted that a proneness to persist, to grow, to spread, and to contaminate adjacent parts, is far more frequently witnessed in the tertiary than in the early formations. I am puzzled to find for mention any other characteristics which distinguish these two classes of symptoms. It may probably be suggested that there is difference as to the parts liable to be attacked, and as to the depth to which ulceration may go; that the symptoms in the secondary stage are all superficial—skin and mucous membranes—and that they do not spread deeply; whilst those of the tertiary stage are apt to occur in cellular tissue, periosteum, bones, viscera, or meninges. I can, however, only admit with great limitation these assertions. Deeply spreading ulceration occurs not unfrequently in the secondary or even in the primary stage; and as to the immunity of the viscera, cellular tissue, and periosteum in the secondary stage, it is far more easily asserted than proven. Some able writers on syphilis have proposed the term "period of gummata" as a substitute for "tertiary stage"—but really gummata may occur in the secondary stage also. It is true that the large gummata of the testis, cellular tissue, liver, &c., are rare, excepting in the tertiary periods, and hence a general impression on the subject, which is, I suspect, very false. But it is not absolutely true that even the large gummata are not seen in the secondary stage. Abundant facts on record disprove such an assertion. Distinctions which relate only to size may easily mislead.

The visceral pathology of the secondary stage might form a chapter in the history of syphilis which has not as yet been written, and for which we possess but few data. It is, however, I feel sure, a great mistake to suppose that there are none to be obtained; and this Society would, I think, be doing a most useful work in encouraging the production of carefully sifted evidence on this point. It is, of course, rare for patients to die during this stage, and

post-mortem examinations are not frequent. The widely spread belief that secondary syphilis, in the acquired form, has no internal pathology worth working at, has probably led to the waste of not a few opportunities which have chanced to occur. I venture to foretell that, when the facts are forthcoming, we shall change our opinions on this point very definitely. There is surely no *à priori* probability in the creed that a blood-disease so severe as syphilis should produce lesions on the skin, in the mouth, and in the eye only; that it should, in fact, affect all the visible parts, and avoid all the concealed ones. The fact that the latter are concealed is probably the only reason why we believe so. The real difference between the secondary and tertiary manifestations, let me repeat, concerns probably some minor facts as to their course and tendencies rather than the parts attacked. In the secondary stage all the morbid processes are transitory, and tend to spontaneous cessation; whilst the tertiary ones are more persistent, and tend to destruction of the parts concerned. But, apart from mere inferential conjecture, there are certain facts already ascertained which seem to discredit the opinion that certain tissues are attacked in the secondary stage, and certain others in the tertiary ones. Time will not permit my mentioning cases, but I may briefly enumerate the following points.

1. In the inflammations of the eye well recognised as early and secondary we have various tissues involved: the iris, which is fibro-muscular; the hyaloid, the retina, and usually, I think, at a somewhat later period, the choroid. In the iris, and in the choroid, little tumours, analogous, I suspect, in all respects to gummata, are seen.

2. When the internal ear is attacked in acquired syphilis, a very rare event, it is usually during or immediately after the secondary symptoms.

3. In two cases of death from syphilitic disease of the heart which have come under my own notice, the event in both occurred in the secondary stage (myocarditis with gumma).

4. In one of the latter cases (examined *post-mortem* by Dr. Sutton) definite gummata were found in both testes and in the spleen, although the secondary rash was still out in the patient's skin. A similar case in an infant, three months old, with secondary rash, has recently been brought before this Society by Dr. Coupland. Parallel facts have been recorded by French observers.

5. It is very common for patients during the secondary stage to complain of symptoms which would imply disease of the same tissues which are attacked later on in the tertiary period. Rheumatoid pains often with slight swellings over bones represent probably the nodes of a later stage. Febrile disturbance, severe headache, loss of appetite and strength, are all symptoms which the eruption on the skin scarcely suffices to explain. Transitory albuminuria is not uncommon, and some authors assert that jaundice may occasionally occur. We should probably see more of such symptoms were it not for the common practice in England to assail the disease with its antidote.

6. The strongest reason for believing in the frequent occurrence of visceral and periosteal lesions in the secondary stage of acquired syphilis is, that they are very common in the corresponding stage of the inherited disease. In the latter the secondary stage is far more severe than it is in adults, and often ends in death, and abundant opportunities are afforded to the pathologist. In infants, it is well known that visceral and bone lesions occur not very infrequently and simultaneously with the skin-eruption. The best example of gummata in the liver that I have ever seen was from an infant. Mr. Canton has described in our 'Transactions' a case in which these growths attained a large size, and showed all the characters which are displayed in those of the adult. The able work of Dr. Taylor, of Boston, just published, contains abundant evidence on the same point. To Wegner, of Berlin, we are indebted for important investigations proving the frequency of periostitis in infants; and when syphilitic sarcocele (gumma, and often large) occurs in inherited disease, it is always in young children and sometimes in infants. Nor is it possible to dismiss this class of facts by suggesting that many infants begin with the tertiary stage, and that probably those who show internal lesions are born of parents who have suffered from the disease long ago. It is, I believe, almost invariable for infants to begin with the secondary stage, whatever may have been that which their parents may have reached. And, further, in not a few cases in which infants have displayed phenomena ordinarily ranked as tertiary, they were the offspring of parents who had but recently suffered, and they themselves showed at the same time the usual symptoms of the secondary one. It is certain, then, that, in infantile syphilis at any rate, visceral and periosteal lesions often occur in the early

periods. Let it be added that precisely the same kind of differences are observed between the periostitis of infancy and the periostitis of some years later, that we see between the transitory bone-pains of the secondary stage in adults and the more lasting nodes which follow years afterwards. In the infant the epiphyses and adjacent bones swell and become tender; but, after a while, all trace of enlargement passes off, and unless, as some times happens to a growing bone, deformity is caused, no trace of the malady remains. Five or eight years later, very probably the same child's bones may become thickened by permanent osseous nodes.

The theory suggested as to the relationship between the secondary and tertiary stages would amount to the abandonment of the latter as any true stage at all. I would emphasise the teaching of those who regard tertiary symptoms merely as sequelæ. Many authors, impressed with the difficulty of arranging in any orderly manner the various sequences of syphilis, have been willing to give up the use of the term tertiary; but it has, I think, always been found necessary to substitute for it some other in no respect more satisfactory. In Lancereaux's able treatise we have the term "period of gummy deposits." My argument is, however, that gummy deposits occur also in the secondary stage, and that in this they differ only from those of the tertiary in that they rarely attain any large size, and usually subside spontaneously. The gummata of the early period are usually small—miliary as they have been called; those of the tertiary sometimes attain a large size. On this admission of fact follows closely an hypothesis which may prove important. Are not the tertiary gummata probably regrowths in structures left behind from the secondary stage? Such an hypothesis, if plausible, would undoubtedly help much to the simplification of the subject; it might also in the future assist in giving definiteness to our plans of treatment. The ingenious doctrine of residual abscesses, ably propounded by Sir James Paget, may aid us in the appreciation of this suggestion. We need not, indeed, go far in any direction of pathological observation for proofs of the tendency of parts once diseased, or the site of morbid products, to relapse even after very long periods of quiet. An especially curious and apposite one is afforded by the rare cases in which indurated chancres relapse. The fact that indurations sometimes recur in parts where formerly chancres existed, without any fresh contagion, has been noticed by several writers, and, amongst others, Mr. Lee

has recorded an example of it. It has, however, I think, scarcely received the attention which it deserves as an illustration of pathological law. I have under occasional observation at the present time several patients who are subjects of the relapsing chancre. Indurations, in all respects like primary sores, occur repeatedly, and without any fresh contagion, and present features which would defy the most experienced observer to diagnose them excepting by the history. They may remain for a few weeks, or even months, and may occur many years after the patient has passed through his secondary stage. Mr. Lee has suggested that they are to be regarded as tertiary gummata; but it is to be noted that they are as hard as cartilage, never slough, often disappear spontaneously and are always located exactly in the site of the original sore. It is this latter fact which seems to me so important in reference to the theory just suggested. It may be well to add that the facts that these relapsed sores are never followed by secondary symptoms, and that they may occur not once or twice, but half a dozen times to the same individual, conclusively support the patient's assertion, that they are not produced by fresh contagion. If an indurated chancre may, in virtue of something which has been left behind, develope afresh in its old site ten years after the primary disease, why may not a like event happen to the cell-products of the secondary stage? It is not to be forgotten that a difficulty may be suggested in reference to this hypothesis, in that the tertiary gummata occur often in parts which are not proved to have been involved in the secondary, and are absent in others in which they frequently are so. For instance, secondary iritis is common, but tertiary iritis or tertiary gummata in the iris are very rare. This objection is probably more apparent than real; and we must always keep in mind that tertiary phenomena of any kind are exceptional; and, further, that it is quite possible that regrowth is the most likely to happen where the original development has been but slight, and the cell-growth has been checked before its power was exhausted. It may, also, be fairly urged that, although exceptions happen, yet, as a rough rule, the tertiary neoplasms are met with in or near the parts which are most commonly affected in the secondary stage. The skin, the subcutaneous cellular tissue, the palate, and the throat, are their more frequent sites. It is true that the brain, nerves, muscles, and viscera claim our attention much more; but this is on account of the greater importance and

interest of the symptoms produced, not because they are really the most frequent sites of tertiary gummata.

In connection with this theory, that the tertiary growths occur in cell structures which have remained over in a quiescent state from the secondary period, it is matter of justice that I should mention the great name of Virchow. Amongst the many suggestions for which we are indebted to his labours, is his well-known theory that relapses of symptoms are caused by stores of poison which have remained latent in various parts, especially in lymphatic glands, and, undergoing development after an interval, cause fresh contamination of the blood. This, however, is not the same idea as that the tertiary growths result, not from blood-contamination in any way, but simply from local renovation of long-resting germs.

In contrasting the course of inherited syphilis with that of the acquired disease, several remarkable features at once arrest our attention. Amongst these are the severity of the secondary stage, (often fatal) and, on the other hand, the frequent omission of all early symptoms; the remarkably long periods of latency which ensue after the cessation of the infantile symptoms; and, lastly, the great rarity of most of the conditions, which in the acquired form we rank as tertiary. I am warned that I must be exceedingly brief in what I have to say on these topics, and to the first two I will not further allude. The periods of latency which are often witnessed constitute, however, a phenomenon too remarkable to be passed over. As is well known, most syphilitic infants suffer during the first few months of life from affections of the skin and mucous membranes which are clearly analogous with the early secondary symptoms in adults. These pass off after a time, and at the end of a year, if the child has survived, it usually appears to be quite well. It may now remain for five, ten, twenty, or even five-and-thirty years without any further indication of its taint, and then may occur some definite and most peculiar affections. That there is a form of interstitial inflammation of the cornea, which when well characterised can be recognised as most certainly due to inherited syphilis, is a fact which, I believe, is not now doubted by any English observers. It is usually symmetrical, and, in a large majority of cases, it runs its course and disappears spontaneously, and as completely as does a secondary syphilitic rash. Nor are the facts as to the long period of latency which may precede it more in dispute than its own character, and I shall scarcely be blamed for

exaggeration if I say that they are amongst the marvels of pathology. Not only may this form of keratitis occur to a grown-up person who has had no symptoms of syphilis since infancy, but it may happen also to one who has never had any symptoms before, and may yet be itself most characteristic. It is difficult to offer any explanation of the cause of this long delay, other than that it has, perhaps, something to do with the gradual development of the tissues. In connection with it we must remark that, however long may have been the interval of latency, interstitial keratitis is still plainly one of the secondary group. Its symmetry and its tendency to spontaneous disappearance both prove its position in this respect, and we note, besides, that certain other phenomena which sometimes occur at about the same age show similar characters. The choroiditis, and the disease of the ear which in these patients so often lead to deafness, are both of them usually symmetrical, and so also very often is the more chronic form of periostitis. I do not mean that the symmetry is absolute. It is not the Dutch-gardener sort of symmetry, which insists on an absolute correspondence, both as to time and features, in the two halves; but still it is sufficiently well marked to assure us of the fact. Very often one eye takes precedence by a short time of the other, and often one suffers more severely than its fellow. I have yet another and a stronger reason to urge for placing the keratitis and the inflammation of the internal ear, which are so frequent under the circumstances described, in the secondary class; namely, that when met with in acquired syphilis they are distinctly secondary. Both are, then, very rare. Of the internal otitis I have seen but few examples, and of the keratitis but one; all the cases, however, occurred to those who had but recently acquired their disease. Until within the last three months I have always been inclined to deny that the interstitial keratitis common in inherited syphilis had any analogue in the acquired disease. There is, however, at present in the Moorfields Hospital, under Mr. Wordsworth's care, a case which, as far as my own observation has gone, is unique. It is an instance of acute symmetrical keratitis in connection with acquired syphilis, and it occurs simultaneously with a general papular eruption, and without doubt within a few months of the primary sore. Thus it would appear to be a fact in hereditary syphilis, that conditions which in the acquired disease would occur within the first year, may be delayed for ten or twenty. Next let us note that the

conditions which chiefly attract attention in the tertiary stage of acquired syphilis are almost wholly absent in the subjects of inherited taint. Large gummata in the cellular tissue, in muscles, in the tongue, in the viscera, or in connection with the cerebral meninges, are almost unknown. The paralysis of single muscles of the eyeball or of those of the face; the attacks of hemiplegia and of optic neuritis, with which we are so familiar in subjects of acquired disease, are, I think, never seen in the inherited form. The able work of Dr. Buzzard on 'Syphilitic Nervous Affections' does not record a single example of such in connection with inherited taint; and although Dr. Hughlings Jackson has with his usual zeal availed himself of almost unequalled opportunities, he has succeeded in getting together but few cases—and none exactly of the kinds I have specified. It is true that epilepsy and some forms of idiocy and insanity, and some other anomalous spinal symptoms, are met with in heredito-syphilitic patients; but, I repeat, we have none of the cases in which symptoms are supposed to be due to gummata, whether of nerve-trunks, of the arterial coats, or of the coverings of the brain.

To the last-named author we owe most of our knowledge as to the discrimination of the different anatomical lesions which cause nerve-symptoms in syphilis. It is mainly in consequence of the elucidation of the subject due to his labours that I am enabled to make the statements which I now record.

It is clear that we have yet much to learn respecting the laws of hereditary transmission. As just remarked, it is exceedingly difficult to assign reasons why phenomena which in acquired disease are crowded together in the first year or two, should in the inherited form be separated by ten or twenty. Nor can we offer any explanation of the fact that in the late periods of the inherited disease there is no tendency to the formation of the neoplasms frequently seen in the acquired form. I said that the chapter describing the internal pathology of the secondary period in acquired disease had yet to be written, and I may now say the same as regards that of the tertiary period in the subjects of inherited taint. With but rare exceptions the latter do not appear to suffer from any maladies which tend to shorten life. I made this statement in print many years ago, and nothing has occurred since to induce me to modify it. Although I have had constantly under observation numerous adolescents and adults well known to have suffered

severely from inherited syphilis, neither in them, nor in their brothers or sisters, have I encountered any special disease of important organs. They have suffered in their eyes and ears, and sometimes in the skin; but here, for the most part, their liabilities seem to end. I do not think that I have made, or known made, more than half-a-dozen *post-mortem* examinations in such subjects, and in these for the most part no very noteworthy lesions were found. In two such death was preceded by long-continued albuminuria, and suspicion occurred that possibly the very long-continued use of iodide of potassium might have had something to do with it. So meagre is our knowledge of this department of the pathology of syphilis, that our Society would, I think, do well to encourage the production before it of all evidence which may in the future be obtainable, with a view to its permanent record in our 'Transactions.'

I now come to the last of the questions which is on my list for consideration, Is there any reason to believe that a taint of syphilis may mix itself with other causes of disease, and produce results of a hybrid nature? It is to scrofula and scrofulous disorders that chief reference is here made. Does syphilis in any way predispose to scrofula or tuberculosis? Vague suspicions have been entertained on this head almost since the earliest date of recognition of constitutional syphilis; and opinions, often of a very sweeping tenor, have been expressed with great confidence by many authorities. Formerly, indeed, it was a question very difficult of investigation. I would venture, however, to submit to the Society that the facts which have been accumulated during recent periods, and the new symptoms which have been placed at our disposal, justify us in believing that syphilis produces in all its stages special and wholly peculiar lesions; and that although these may easily be mistaken for struma, they have in reality nothing whatever to do with that state of constitution. It is undeniable that syphilis, and sometimes the treatment required for it, may for a time enfeeble the system, and that during this state of debility a person predisposed to phthisis or struma may experience an increase of his proclivity. In this, however, there is nothing more than what is common to all causes of enfeeblement, whether special or otherwise; and I do not think that in the instance of syphilis it amounts to much. Syphilis may be contracted by persons of the most various states of health, and by those who are the subjects of special diathesis.

Strumous or tuberculous subjects, those who suffer from gout or from psoriasis, and those of extreme enfeeblement of circulation, may all in turn present us with examples of this specific disease. As a rule, we do not observe any modification of the one by the other. A common psoriasis rash may persist during the treatment of constitutional syphilis, and may remain for years afterwards, just as it did before ; and the same is, I think, true in the general way of lupus, leprosy, and other chronic maladies which show their chief symptoms in the skin.

In investigating the relationships between scrofula and syphilis we might conveniently take as type-examples of strumous manifestations two common and fairly definite maladies,—ulcers in the cornea, and Lupus. We have chiefly to consider the hereditary form of syphilis. A large majority of examples of chronic ulceration of the cornea (strumous ophthalmia) occur in those who show no signs of hereditary taint, but who, either in themselves or near relations, betray a tendency to other forms of scrofula. When such ulcers happen to those who are known to be the subjects of inherited taint, for the most part they run their usual course and require their usual remedies, while syphilitic keratitis is the same malady in the strumous and in the healthy. I do not say that this is invariable, but I think it is the rule. Respecting the common and interesting disease of the skin known as lupus, grave suspicions are still entertained by many who are well informed. To my own mind, however, the evidence is clear. True lupus has nothing whatever to do with syphilis either acquired or in the first or second generation of inheritance. The evidence on this point is of two kinds. In the first place, we hardly ever, as far as my experience goes—I might, I think, say never—meet with common lupus in those who are obviously the subjects of inherited taint. It must be granted, however, in the fullest manner that not all, or nearly all, of those who really inherit a taint betray it either in physiognomy, teeth, or by concurrent disease of suspicious character ; and, further, that it is precisely in those who do not so betray it that we must expect the history of symptoms in infancy to be wanting. In these cases, however, it is seldom that the patient is an only child, and it is very rare for a whole family to escape. If the surgeon will widen his inquiry, and, instead of contenting himself with the original patient, inspect carefully all the brothers and sisters, he will generally find evidence that is conclusive. In this way the

recognition of signs of inherited taint in one individual very commonly reveals its existence in possibly a quite latent form in three or four others. We identify not merely an individual but a whole family, and thus very much extend our area of observation. Making use of the symptoms of physiognomy, teeth, keratitis, and choroiditis, I do not think that it would be difficult in any large out-patients' institution to soon make the acquaintance of several hundred individuals concerning whom the observer might be sure that they all, in greater or less degree, inherited a taint of syphilis. Any one who had done this would be in a position to say whether lupus occurred in more than average proportion amongst this class; and I have no doubt as to what his verdict would be. It is difficult to leave this part of our subject without suggesting that most valuable work in this direction, not only as regards lupus and scrofula, but in reference to the late results of inherited taint, might be done by those of our members who have the advantage to be engaged in family practice. If all the cases in which family advisers are aware of the fact, either of the existence of syphilis in parents or its actual presence in offspring, or both, would carefully note all that occurs to all the children in such families, not only in childhood, but in after life, a mass of facts might soon be collected which would help greatly to the solution of many important questions. It is far more difficult to follow out these inquiries in hospital practice.

There is yet another line of argument as regards the non-connection between syphilis and lupus. We have said that lupus does not occur frequently in the hereditarily syphilitic, or their brothers and sisters. Next, I assert that amongst those who have lupus and their relations, you will find, as a rule, not the slightest reason for suspecting specific taint. No one can, I think, doubt for a moment that lupus may occur under conditions which make the existence of syphilis either in parents or grandparents most improbable, nor, indeed, that it is, for the most part, under those conditions that we usually meet with it. Now, as lupus is a definite and peculiar malady, the probability is that it has a definite cause, and it is not due in one case to the tubercular diathesis, and in the next to that of syphilis.

There is no doubt that syphilis may produce skin-diseases which superficially very closely resemble lupus. This occurs most frequently in the acquired form of the disease, and in a few of these

the diagnosis is really very difficult. The essential difference in nature is, however, usually proved at once by the results of treatment. In inherited syphilis we seldom or never see anything which at all resembles the tubercular or common form of lupus. We do meet not unfrequently, however, with a disease which formerly was called phagedænic lupus, but which, like other varieties of phagedæna, is always and wholly the result of syphilis. It spreads far more rapidly than true lupus, and is easily curable by measures which would avail but little against the latter. Above all, it is easily distinguished after cure by the fact that its scars remain perfectly sound without the least tendency to the relapses which are so constant after cures of lupus.

Here, Mr. President, I must draw to an end. In attempting to fulfil the responsible duty with which the kindness of your Council has honoured me this evening, I have thought it better to avoid all subjects of mere detail, such as might seem to be more appropriate for special communications. I have kept two points chiefly before me: to try, if possible, to simplify and make more orderly our general view of the subject, and to direct attention to those parts of it which seem most to need further investigation. To my great regret I have, under compulsion as to brevity, found but little opportunity for mention of the names of those to whom we are indebted for the facts and suggestions of which I have freely availed myself, and of which, indeed, my paper is in some sort a running summary. Our knowledge of syphilis has, indeed, been "set in the light of many minds." If we were to pass over in silence all excepting those who have contributed recent additions to our stock, and of them take only those who have worked at the departments most interesting to this Society, the list would still be very long, and the difficulty of doing justice very great. In France, Ricord, Diday, Lagneau, Lancereaux, and Fournier; in Germany, Virchow, Wegner of Berlin, Dittrich of Prague, Von Bärensprung, and Zeissl; in Scandinavia, Bergh, Boeck, and Bidenkap; in America Bumstead—have taken foremost places; but their names do not even tithe those who have a claim on our gratitude. At home Dr. Wilks, I believe, led the way as regards visceral syphilis; and the earliest contributions were most of them made before this Society. Dr. Murchison, Dr. Moxon, Dr. Weber, Dr. Payne, have also contributed most valuable facts.

Our knowledge of the diseases of the nervous system, to which I believe Dr. Reid, of Belfast, was the first of our countrymen to contribute, was early helped on by Dr. Meadows, who, from the practice of the late Dr. Todd, published some cases in the 'King's College Hospital Reports;' and more recently by Dr. Hughlings Jackson, Dr. Buzzard, Dr. Russell, and Dr. Broadbent. Nor must I omit to mention, as zealous and successful workers at the general subject, the names of Lee, Berkeley Hill, De Méric, and Gascoyen; nor, last, the author of the most recent and, I think, most comprehensive and able treatise on the disease that has yet appeared—Dr. Bäumler, whom we would gladly claim as, at any rate in training, half an Englishman.

The regulation of the discussion which is to follow, and the decision as to the precise scope which it shall take, will, I am glad to know, rest in much abler hands than mine. That I may not, however, fail of any part of my duty as its introducer, I have endeavoured to epitomise under the following heads some of the chief points raised in my paper. It will be seen that they are by no means closely connected; and that it will be quite competent for any speaker, if he prefer, to confine his attention to a single one, upon which he may be in possession of special facts, without adverting to the others.

Are there any facts which favour the belief that syphilis continues to be a blood-disease after the cessation of all tendency to produce symmetrical symptoms?

The dearth of facts illustrating the internal pathology of the secondary stage of acquired syphilis.

The importance of all facts or arguments for or against the belief that the gummata of the tertiary stage are purely local, and result from renewed growth in formations left over from the exanthem stage.

The value of facts as to the pathology of the late periods of inherited syphilis.

Mr. H. Lee.—I believe, sir, it is a well-known fact that, when the sucker of a pump is a little dry at first, by putting a very little water in it you may bring a great deal out. In obedience to your command, therefore, I will make one or two short observations. In the first place, I quite concur with you, sir,—that is one point on

which there will be no discussion or division of opinion,—in thanking Mr. Hutchinson most sincerely for the scientific, careful, and talented way in which he has brought forward these several points. The area over which he has travelled is so large, and the number of subjects he has introduced is so great, each one of them being sufficient to occupy an evening in discussing it, that I think we must adopt his suggestion and consider them separately. The first point that struck me was that which Mr. Hutchinson first introduced about the blood-disease. Now, if we limit the term blood-disease to the stage to which he wished to limit it, certainly we want some better definition of a blood-disease. Mr. Hutchinson has described gummata in the liver at the same time that there were secondary eruptions on the body. The secondary eruptions he acknowledges to be a blood-disease, and we cannot but suppose that the gummata depended upon the same. We cannot think that the gummata depended upon one thing and the eruption upon another. At a subsequent part of his paper he describes gummata in a child's liver, and that, he says, is not a blood-disease. In the one case it would come under the head of blood-disease, and in the other case it would not be a blood-disease. I think, if I caught Mr. Hutchinson's observations aright, it was during the state of inoculability that he thought it was a blood-disease; but I cannot at all agree with that view. I should certainly think that if a father transmitted the disease to his son, that father must have had the disease in his blood in some way or other. Therefore I should consider all hereditary diseases as emanating from a blood-disease. Another point of considerable interest was touched upon by Mr. Hutchinson; that is, the way in which new matter is deposited principally in primary, but also in secondary syphilitic affections, and the way in which it is removed. Mr. Hutchinson classes all syphilitic affections under one head as derived from one poison; but I should have liked very much to have something more definite as to why one result is produced in one case and another result in another; whether it is something about the kind of inflammation that produces the inoculable product, or whether it is that in one case and something else in another. But, with the exception of phagedænic ulceration, Mr. Hutchinson, I think, has not given us any information upon these points. Certainly there are three distinct forms in which we can see the results of syphilitic inoculation develop themselves. There is an inoculation where we see new matter deposited perfectly

circumscribed, accurately defined, which does not tend for a long time, at least in a healthy state of the body, and perhaps not at all, to undergo ulceration or suppuration. That we call distinct infecting inoculation. In another case we see that instead of that the inoculable point suppurates at once, ulcerates at once; there is no new matter deposited, and there is always a loss of substance, which there was not in the first case. There is a third form of inoculation (here I think I touch upon somewhat new ground) in which solid new matter is deposited which has not the characters that I mentioned in the first case, but it gradually diffuses itself into the consistency of the surrounding textures and has a tendency quickly to degenerate. That is what I think Mr. Hutchinson calls his soft sore. These are three very distinct forms of action, and they are not only perfectly characteristic, but, I believe, diagnostic of different affections. The first is the real infecting sore, which indicates the general infection of the patient's system; the second is the suppurating sore, which always leaves a loss of substance, which always suppurates and ulcerates, and, as far as the evidence goes which I have been able to obtain, never affects the constitution; then comes the third form, which has produced the confusion between the other two, where a person, having already had syphilis, is re-inoculated with that syphilis, and the induration has not then the period of incubation which is natural to syphilis acquired for the first time, but developes its results at once, and the newly deposited matter rapidly degenerates; this form of disease afterwards simulates the soft sore; but it is not a soft sore; it has not run the same course; it has had the solid matter deposited first before any ulceration takes place. In the first there is solid matter only, as the essential part of the disease; in the second there is a loss of substance only, as the essential part of the disease; in the third there is a combination of both; and I think that, from not recognising this third form of syphilitic inoculation, the confusion between the other two has arisen.

Dr. Charles Drysdale.—So many points have been touched by Mr. Hutchinson that I can only allude to one or two. With very much that he has put forward I entirely agree, but one or two of his assertions seem to me to be capable of critical examination. For instance, the assertion that *Dualism* as a doctrine was dead was puzzling to me. If it had meant that the views of

Carmichael, of Dublin, that there were several varieties of the syphilitic virus, were no longer tenable, I should have agreed entirely to that; but he evidently spoke of the idea of the soft chancre being distinct from syphilis as being a defunct doctrine. To this I entirely demur. At any rate, both in London and in Paris, there are very many distinguished persons alive at this moment who are quite convinced that soft sores are not a part of the syphilitic drama at all. Thus, the hard sore has a long incubation, sometimes a very long one. I had a case the other day in the wife of a sailor who married in July, and whose husband left her in August, but whose sore did not appear until November, or three months, at least, after she was infected; whereas the soft sore has no period of incubation at all, as those know who have tried many inoculations, or have been present at Professor Boeck's wholesale inoculations, performed some years ago in London. The two diseases seem to me to be quite distinct, and, curiously enough, they are, at this moment, not often met with together in Paris. Thus, I have in my pocket a letter from Dr. Charles Mauriac, physician to the Hôpital du Midi, who informs me that, whilst hard sores and syphilis are more prevalent than they were ten years back in Paris, the soft sore is sometimes not seen at his hospital for weeks. I submit that, if Mr. Hutchinson be right, and the soft sore is only a product of syphilis (which he has not this evening proved), this fact mentioned by M. Mauriac would be hard, indeed, to account for; whereas if we believe, as I do, that the soft sore has always existed throughout history (for it was described by Celsus), and that the hard sore is a recent introduction in 1493, there is nothing mysterious if they should often coexist. I am inclined, with Mr. Hutchinson, to see a very great likeness between syphilis and the exanthemata; indeed, in some cases of syphilitic roseola I have noticed a fever with a temperature of 104° F. The incubation of the poison makes syphilis like smallpox; but the eruptive period of syphilis usually lasts about a year, or may even extend sometimes to twice as long as that, in very rare cases. I am almost inclined also to be seduced by the idea of a parasitic origin for the disease, as Mr. Hutchinson believes, and with this notion, and moved by his opinions and those of Dr. A. Fournier, of Paris, I have recently given small doses of green iodide of mercury for long periods in the secondary stage (one third of a grain twice daily for perhaps some ten months) with the idea of thereby

attacking the virus in the tissues, and thus warding off the tertiary stage. There are, however, cases which puzzle me greatly, for I have published one where a woman had no less than eleven dead births, one after the other; and in other cases syphilitic children seem to have been born more than five or six years after infection, which certainly does not look as if the virus *germs* had then left the system. I think that the mother of a syphilitic child has *always* contracted the disease in her system; although experience shows that sometimes syphilis is such a mild disease that scarcely any symptoms of it may be seen. The ordinary rule, however, is that a mother, in two or three years, begets quite healthy children again, as if the germs were then all dead. Women with even fatal forms of tertiary lesion sometimes give birth to fine healthy children. One woman who attended the Metropolitan Free Hospital for many years with sloughing tertiary sore throat, and eventually died of it, gave birth to two fine children, which had no apparent taint of the disease. In the secondary period, again, gummy deposits are not so rare as used to be thought. They are found in the testicle about the end of the first year; in the liver and in the brain, in some cases very early in the disease. A man came to my hospital with right hemiplegia and aphasia, some years ago, whose body was completely covered with the rash of syphilitic roseola; and I have several times seen enlargement of the liver with jaundice, whilst roseola was present. Another difficulty in admitting that the tertiary period is entirely free from the germs is that, occasionally, in traumatic cases, as shown by M. Verneuil, of Paris, rupial eruptions may arise very many years after the original infection under the influence of some operation. Again, nodes certainly appear on the forehead sometimes two or three months after contagion. But, as a practical rule, I confess I always tell patients with syphilis that they may marry without much fear of contaminating their children in a couple of years after the mucous tubercles, &c., are over, *i. e.*, about three years after primary infection.

When children get syphilis, their disease, in my experience, is usually mild when not inherited; such children do not often seem to suffer much from tertiary disease. With regard to Mr. Hutchinson's splendid discovery of the way of diagnosing inherited syphilis, by means of the two upper central incisors, it is a singular fact that, in London, this is now deemed by experts as clear as the

sun at noon-day, whereas, in Paris, many great syphilographers have their doubts about it; for instance, MM. Verneuil and Fournier. The place to see such cases is in a large hospital for eye-diseases, such as Moorfields. Nervous diseases are, I think with Mr. Hutchinson, very rare in inherited syphilis; but bone disease and other tertiary phenomena, with affections of the tongue, are not so very uncommon, and patients with inherited syphilis cannot, I think, often live to a very old age. Hereditary syphilis is quite distinct from scrofula, but syphilis causes a kind of pulmonary phthisis with cavities in the lungs curable by iodide of potassium. Women with hereditary syphilis give birth to healthy infants. I examined the scrofulous children at Margate and at Berk-sur-mêr, and saw no cases of hereditary syphilis among them. With regard to the treatment of the various periods of syphilis, experience has shown me that late on in the disease mercury is usually of scarcely any use. In tertiary sore throats, ozœna, and sloughing ulcers, large doses (10 grains or more) of iodide of potassium should be given. In rupia both iodine and mercury may be tried. It is only in forlorn-hope cases of tertiary disease that mercury need be given, in my opinion. Mercury, in short, for the germ period, iodine for the tertiary lesions.

Adjourned Discussion, February 15th.

Sir James Paget.—I desire to begin by expressing my agreement in the main with the doctrines of syphilis which Mr. Jonathan Hutchinson propounded at the last meeting of the Society. I agree with him in the belief, if he will allow me to quote his own words, that in syphilis we have to do with but one malady and one virus; that in syphilis we have to deal with a specific fever of prolonged definite stages, and that it is the sequelæ of the fever to which we give the name of tertiary symptoms.

Mr. Hutchinson has rightly indicated some of the points to which he would suggest that the discussion of the subject should lead; and I will take one of these, namely, the relation of the tertiary symptoms which he regards as the sequelæ of the syphilitic fever, if I may use the term. In this view I do not pretend to add any

new facts to those which Mr. Hutchinson has himself stated. If I can add anything to strengthen the arguments he has adduced, I think it can only be by pointing out the very close resemblance which exists between the tertiary symptoms and those which may be regarded as sequels of other forms of fever. The very characters which Mr. Hutchinson has pointed out as indicative of the tertiary period, the sequel of the syphilitic fever, are, first, that the symptoms which it presents are not symmetrical; then, that it is a disease which is not contagious, nor yet, so far as experiments have yet proceeded, distinctly communicable. Moreover, I suppose it may be added that it is very seldom transmissible by inheritance.

Let me take occasion to speak personally of my gratification that the doctrine of the symmetry of disease is upheld upon so strong grounds as those which Mr. Jonathan Hutchinson has introduced; for, so far as I know, the full significance of symmetry as characteristic of a blood-disease has scarcely been dwelt on since it was the subject of papers communicated to the Royal Medical and Chirurgical Society by Dr. William Budd and myself; and if, in speaking of syphilis as a blood-disease, Mr. Hutchinson has raised some strong questions in modern pathology as to whether the term blood-disease can, under any condition, be justly used, I will recommend those who object to the term to read carefully the paper by Dr. William Budd in the twenty-fifth volume of the 'Medico-Chirurgical Transactions.' I think they will find there something as near to demonstration as can be arrived at in pathology, that diseases which are symmetrical owe their existence to a morbid material in the blood; and that those which are regarded as the morbid products of the disease shown symmetrically are really the morbid material combined for a time with the natural texture of the parts affected. To that doctrine I still hold; and I may have occasion to refer to it in that meaning many times in what I say. The years that have passed—more than thirty now—have shown, doubtless, that the nervous system has a larger share in the determination of symmetry in disease than either Dr. William Budd or myself at that time supposed. Nevertheless, the main facts hold true, that, as Mr. Hutchinson has illustrated, a symmetrical disease does, by its symmetry, indicate the presence of a morbid material in the blood. Moreover, I would add to what he has himself demonstrated, that the product or, as we call it, the

appearance of the disease, is due to the combination of the morbid material with the natural textures of the parts.

Mr. Jonathan Hutchinson asks, as one of the first questions which he submits for the consideration of the Society—if I may again use his own words—Are there any facts which favour the belief that syphilis continues to be a blood-disease after the cessation of all tendency to produce symmetrical symptoms? Now, I think, that to answer that question we must inquire what are the characteristics of the sequels of other fevers in reference to symmetrical disease. It is clear that the sequels of all other fevers are, with very rare exceptions, not symmetrical. There are the sequels of pyæmia, the pyogenic fever; its phenomena, in its acute stage, are rarely symmetrical, but when it passes into its sequels in the chronic stage its phenomena are perhaps still more rarely symmetrical. They indicate a singular similarity to the tertiary symptoms of syphilis, in that they very commonly affect always the same textures; as, for example, a large number of them are shown only in suppuration in the connective tissue; or in other persons they appear only as slowly successive instances of necrosis, herein indicating one of the most marked features of resemblance between the sequels of the best marked acute fever, and of that which some may deem the least marked—the syphilitic fever. But they are not symmetrical.

Again, intermittent fever is a fever well to be studied in its likenesses to syphilis, because it passes even beyond syphilis in the duration in which it will hold itself, as I suppose, in the blood of the patient. Thirty years are referred to as the time that syphilis may continue without intervening symptoms. It is no uncommon thing for ague to be repeated in its phenomena thirty years after the time of its first occurrence; but it is not notable that what may be regarded as the sequels, or the latest symptoms, of ague, are not symmetrical. They are best of all marked in the various neuroses, which are recognised under the terms of brow-ague, migraine, and others, for the most part not symmetrical affections of the nervous system.

Then I may instance the sequels of typhoid fever, a subject which, I venture to think, has not sufficiently attracted the attention of physicians, probably because the cases more frequently fall into the hands of surgeons. Within some years past I have been struck with the great variety of surgical sequels of typhoid fever,

and amongst them I have most rarely indeed met with instances of symmetrical disease. Among the instances of necrosis as the sequel of typhoid fever, ten or twelve in number, I have seen only one symmetrical—a well-marked case of symmetrical necrosis of the lower ends of both tibiæ; but in all others, whether affecting the tibia, the ulna, cranium, or other bone, there has been always an absence of any sign of symmetry. So in the case of phlebitis, which is common in the femoral vein as a sequel of typhoid fever, I have never seen symmetrical phlebitis. Nor have I seen a single instance of symmetrical affection of the nervous system as a sequel of typhoid fever, except in one or two cases, where complete paraplegia was produced; but in all those which are more common, paralysis of the anterior muscles of the leg, or of part of the muscles of the arm, or any other, I have not yet met with one instance of symmetrical paralysis. Lastly, with regard to an affection which is not unfrequently seen as a sequel of typhoid, suppurative periostitis of the ribs, six or eight of these cases that I have seen have shown no sign of symmetry.

Therefore, I think we may hold that in this absence of symmetry in these sequelæ, that is, in its tertiary symptoms, the syphilitic fever, if I may so call it, shows a remarkable feature of resemblance with all those other more acute fevers in which we can trace distinct sequels. And in this I suppose it only falls in with what is generally true concerning symmetrical diseases, or diseases of the blood, that in their later periods of existence they all become irregular, that is, they not only lose the regular character of symmetry as affecting both sides of the body alike, but they lose many of the regular characters of form and size and external appearance with which we are familiar. For example, if we watch lepra, or psoriasis, perhaps the best marked instance of symmetrical eruption, if after a time it spread from the limbs over the trunk, or from the upper extremities over the face, in its later periods it commonly ceases to be distinctly symmetrical. Or again, if I may cite an instance of what is not properly, I believe, to be called a blood-disease, yet is distinctly symmetrical (that is, the so-called pityriasis versicolor): it is symmetrical in all its early stages: when it is declining, it does not decline or fade with symmetry; or if it relapse, it does not relapse with symmetry. So with rheumatic gout or with cancer, in the condition in which I suppose, even in this room, it will be generally understood to be a blood-disease,

when it has thoroughly infected the blood in its later stages, and appears in multiple forms: these are not commonly symmetrical.

Therefore I think the rule may generally be, if we are to answer Mr. Jonathan Hutchinson's question as to whether there be indications that in the absence of symmetry a blood-disease has ceased, that the cessation of symmetry as a characteristic of syphilis is exactly accordant with its absence in the sequels of other fevers, and generally with its absence in the periods of decline of nearly all diseases that begin by being symmetrical; in other words, in nearly all blood-diseases. And I apprehend that the reason of this decline may be assigned not only, as Mr. Hutchinson assigns it, to the influence of antidotal remedies, but, besides, to the constantly increasing prevalence of the natural nutritive forces over the morbid ones. For we may certainly hold that syphilis is a disease which does not usually tend to a continual increase and fatal termination; that it is constantly exposed to the strife with the natural nutritive forces, which will not, indeed, simply diminish it in intensity, but, if I may so speak, gradually break it up by making it irregular; and amongst the first signs of its irregularity make it not symmetrical.

But to revert to Mr. Jonathan Hutchinson's question, whether the absence of symmetry may be taken as the indication of the arrest of blood-disease, I must observe that in the hypothesis which follows in his paper, in which he assigns the occurrence of the tertiary symptoms to changes of structure ensuing in the residues of the secondary deposits, he would have to admit that in those residues the syphilitic disease shows itself as secondary, and therefore, as it should be, symmetrical; and he would then have to explain why, being symmetrical in the forms of the secondary period, it becomes unsymmetrical in the manifestations of the third. If he will let me venture to improve his hypothesis, I would suggest that those of which he speaks as the residues of the secondary period are only such as are formed in the period of irregularity which follows the secondary and precedes the tertiary: a period of irregularity in which we should expect that, after the common manner of all blood-diseases, symmetry would cease to be observed, or would, at least, be gradually less and less distinctly observed. In that case I can fully adopt his hypothesis of the deposits which reappear in the tertiary period being produced in the secondary.

But as to whether the tertiary symptoms may be regarded as not due to blood-disease because they are not symmetrical, we must look to further characteristics for decision; and I would venture to believe still that syphilis, so long as it exists in any manifestation at all, is a blood-disease. It may be supposed that the secondary deposits, as the results of blood-disease, may be renewed into disease by any external cause; but this would hardly explain several features of tertiary syphilis. First of all, we can scarcely understand local diseases to be entirely of spontaneous origin; yet the outbreak of a tertiary syphilitic attack is to all appearance as spontaneous as anything that we see in disease. Then, in local diseases, we commonly suppose that the evidence of disease will bear some kind of proportion to the force with which it has been produced; and yet certainly tertiary syphilis in great severity may be produced or made manifest by an external force very small in amount. Then, again, most of the tertiary syphilitic symptoms have their distinct or, as they are commonly called, specific, forms; manners of nutrition, methods of disease, by which they are sufficiently distinguished in our diagnosis from anything that could fairly be called common disease. We can usually distinguish a syphilitic ulcer of the tertiary period from any ordinary ulcer. Very commonly we can distinguish a syphilitic joint from a rheumatic, or a gouty, or a simply inflamed joint. Very commonly, too, we can distinguish a syphilitic periostitis from a common one.

Then, lastly, as another sign of specific character, and therefore, so far, probably of persistent blood-disease, we can discern that, in certain cases, the tertiary syphilitic disease requires specific treatment. I entirely agree with the ingenious expression of Mr. Jonathan Hutchinson that it may be local treatment which we address to a disease, even when we put the remedy into the blood; that we only—if I may so say—use the blood that it may carry the remedy into the substance of the diseased part; and yet it is plain that there are certain tertiary syphilitic diseases accessible to external treatment, as well as those which would be considered internal, but not curable by external treatment. The annular and crescentic ulcers of the tertiary and syphilitic periods are, so far as I know, not curable by any external treatment. We may bring iodide of potassium, or mercury, or any other treatment, to their surface, and give it every opportunity of reaching their very substance, but we do not thereby cure them; we need for the cure of

them, as much as for primary syphilis, either iodide of potassium for their temporary cure, or mercury for their final cure.

Therefore, out of these facts, I should answer, so far as I can, Mr. Hutchinson's first question, by holding that, to the last, so long as there can be any evidence of its existence, syphilis must be regarded as a blood-disease.

I come, next, to that to which I have already adverted, the hypothesis—a most ingenious one, and one in which I am nearly sure there is some real value—namely, that the gummata of the tertiary stage result from renewed growth in deposits left over from the exanthem stage; that, if I may express it otherwise, the exanthem stage of syphilis leaves behind it certain residues in which the disease may be renewed by any casual external conditions. I cannot cite instances of this from my own observation in syphilis; but instances may be cited in general pathology and from common diseases which, at least, will go far to render it not improbable that this may be a true hypothesis, even although there may not be discernible, in the parts which become seats of the tertiary syphilitic disease, any change of structure whatever. I published, some years ago, an instance which occurred in my own person, to show how a residue of disease may be left unobserved, and yet ready for outbreak upon any fit occasion. I had applied leeches to my wrist for a sprain of it, and four or five years elapsed with no trouble whatever in the scars, except that I could count them on my wrist. At the end of five years, when the scars might have been deemed to be as sound as any other texture of the body, I was for the first time exposed to the heat and other conditions of travelling in Italy, and then at once every scar inflamed and ulcerated; and so it occurred for every year in succession when I was exposed to the same influences; every scar and every leech-bite inflamed and suppurated again, and it took four years of similar exposure to those causes of disease before the scars settled down into a condition in which they could resist these influences.

And, if I may tell another personal experience, it will bear with some force on the question of the necessity of studying residues of disease. Four years ago I had an attack of acute erysipelas following dissection-poison. It affected only my left arm and part of my left side, the right arm being wholly unoccupied by it. My left arm recovered, as I supposed, without a blemish in it. For years I went on without the smallest consciousness that there was anything

in that arm which could be affected by any force which would not equally affect the other arm. This winter—I hardly know why—I took cold baths instead of warm ones, and, on every application of cold water to the arm, in which, let me repeat, I am wholly unconscious of any difference from the right arm, there ensues lividity; there is a marked difference between the left arm and the right; such a difference as would spoil the symmetry of any constitutional disease with which I may be affected; such a difference as might be marked by a greater tendency to production of disease in that limb than in the other; a clear residue, not discernible by any other means than those applications of cold; a residue very similar to that which may occur after any specific disease to which any man may be exposed. I am disposed to parody the well-known expression, "Let no man be accounted happy until he is dead," by "Let no man be accounted healthy until he is well examined after death."

There is only one point more in Mr. Jonathan Hutchinson's paper to which I will refer, and that is one in which I feel bound for the first time, not materially, yet in a measure, to differ from him. He enters upon this question—one of the greatest interest—in reference to the relation which syphilis bears to other diseases, and asks, Does syphilis in any way predispose to scrofula or tuberculosis? In answer to this, I should agree with him absolutely that it does not so predispose in any other sense or degree than any other fever does. Syphilis, by long-continued illness and deterioration of health, may let in scrofula or tuberculosis; but so may rheumatism, so may gout, so may typhoid, so may any other disease which, to use the plainest words, in any degree impairs the health of the person disposed by inheritance to scrofula or tuberculosis. Then he speaks of the relations of lupus to syphilis. And there, too, I agree with him wholly that lupus is in no proper sense a syphilitic disease. It is definitely a disease of tuberculosis. I doubt whether any persons the subjects of lupus can be found in whom there might not be traced a well-marked inheritance of tuberculosis; and, if there be a syphilitic lupus, I should hold that it can be found only in persons who become syphilitic after having inherited the constitution out of which lupus is likely to occur; but, when Mr. Hutchinson says that, as a rule, we do not observe any modification of one by the other—referring there to syphilis as it may occur in strumous or tuberculous people, in

those who suffer from gout or psoriasis, and those who have extreme enfeeblement of circulation, I cannot but differ from him. I think that one of the things we have most to study, both in the pathology and treatment of syphilis, is the modifications which it undergoes in persons of different constitutions in whom it may be inserted.

I should hold this even in regard to the primary syphilitic sore. Agreeing with Mr. Hutchinson that there is but one syphilitic virus, I yet cannot but feel that we have to explain the singular variety of forms in which that virus, or the pus in which it is contained, produces its effect in different persons. We may suppose that in some persons the pus is inserted without the virus, and in others not; but it would be an extreme difficulty to tell any means by which the virus can be separated from the pus. I think we should look for facts which would indicate that, out of the constitution of each person into whom the syphilitic virus is inserted, there may come a different—not essentially different—but a modified, result. The facts, indeed, are like those which may sometimes be discerned even now in the insertion of the vaccine virus, to which some are less and others more subject, and some absolutely indifferent, or such affections as we have on record from the old histories of inoculation, where the disease produced was the same in kind, but very far from the same in degree, in all persons in whom it was inserted. So, too, in the secondary period, I think we may see, however obscurely, that there are differences due to the differences of the constitutions of the persons in whom the secondary disease occurs: but much more in tertiary syphilis, in that period in which the syphilitic virus seems to be losing its power or intensity, and the natural constitution of the person comes to have more and more share in the general product of the disease.

I would not venture to call the disease that may occur in a scrofulous person, become syphilitic, a hybrid one: and yet, perhaps, the term is not altogether wrong; but, at least, I would call it a mixed disease, and hold that syphilis inserted in a scrofulous person will, in its tertiary period, produce signs which it may be very hard to distinguish from scrofula—signs in which the characters of scrofula and of syphilis are mingled, and—which is very important—which require that the treatment of scrofula should be combined with the treatment of syphilis, in order to produce a fully successful result. So, too, in gouty persons, and in the rheumatic

and the neurotic, I think we can discern mingled characters of syphilitic disease with the diseases to which their constitutions make them liable; and I think I can be sure, too, that all these several persons require for their treatment that we should have in view not only the specific disease but the constitutional disease with which it is mingled; so that the treatments for the natural constitution and the acquired constitution may be combined.

And this is quite confirmed by what we observe in the sequels of other fevers; for clearly, when we watch the sequels of typhoid fever, we have to explain why one person has phlebitis, another necrosis, and another suppurating disease of a rib; and I believe that those differences may be explained in discerning different constitutional or even different local peculiarities in the persons affected. I am acquainted with an instance in which five members of one family, having suffered from typhoid, have all had affections of the nervous system as its sequels; and the worst case I have yet seen of crural phlebitis following typhoid is the case of a gentleman in whose family I know of four other persons all suffering from disease of the veins of the lower extremities. These, it may be said, are instances in which the sequel of the disease falls only on a part which may be considered weak; nevertheless, that which is true of a part would also probably be true of the constitution; and, at least, we have to keep in mind that, among the many diseases which we have to study, few are so variable in their phenomena, taking them altogether, as syphilis.

Now, we have cast aside—at least, I have, with Mr. Jonathan Hutchinson—the belief in the multiplicity of poisons. But, if there be one poison only, how is it that that one poison produces so many various effects. We cannot ascribe it to the various external conditions in which the patients are placed; neither can we reasonably ascribe it to the variety of treatments; for the treatment of syphilis, as now carried on, is, for the most part, very similar in all cases. It seems to me that the only thing to which we can fairly look is to discern the differences produced in the one disease by the variety of persons in whom it is inserted—the variety of soils, if I may so speak, in which it has to develope and to grow.

These are all the observations that I would make in regard to Mr. Jonathan Hutchinson's paper; but I would not sit down without expressing my admiration of the paper as a whole, and of the thorough, profound view which Mr. Hutchinson has taken of

the whole subject. I venture to make myself proud in calling him one of my pupils. Not my pupil in syphilis : with regard to which I say nothing, but that, at least, I did not interfere with his studies while he pursued them with myself in the out-patients' room of St. Bartholomew's Hospital, where I saw nothing but confusion, and he saw light coming. And I venture to say, if any of us think that in this instance Mr. Hutchinson is looking into outer darkness we shall be very unwise if we neglect his signals when he says that light is come.

Dr. Wilks.—Having been asked to say a word or two on the present occasion, I rise to do so, having always felt an intense interest in the subject of syphilis ; I am afraid, however, that I can throw very little fresh light on the matter, my views being much in accord with the general doctrines laid down by Mr. Jonathan Hutchinson. Permit me, in the first place, a little ebullition of feeling in this matter. When I remember it was only a few short years ago that the subject of syphilis was discussed in this Society in the crudest possible manner, and when I think of the philosophical address which we have had presented to us, I cannot but feel that more has been learned of the nature of syphilis within the last twenty years than was known during the four preceding centuries ; and I cannot but think that the Pathological Society of London has had a great share in this advancement. The younger members of the Society are, perhaps, scarcely aware how we had to fight this question of visceral syphilis in the Society. Being much interested in the matter, and thinking it over this morning, I took the opportunity of referring to a medical journal to see how very recent the modern doctrines were. In a so-called review of my 'Lectures on Pathological Anatomy,' extending to about twelve lines, one of the leading journals said : "We cannot but think that he errs in laying down as law what is considered by most authors still sub judice. For example, syphilitic fibroid deposits are mentioned as of frequent occurrence and of unquestioned character. Now, most of those who have paid attention to the subject regard the proof of this statement of Dr. Wilks as anything but convincing." Taking the intense interest that I do in the advancement of my profession, if I were asked in what direction progress has been made in it, I should be vain and conceited enough to say in the direction of pathology ; and if I were asked in what par-

ticular department, I might mention that of syphilis, and I should point to this Society as having had a large share in the work. The younger members have brought, night after night, specimens in the despised soup-plates, endeavouring to improve our knowledge, until we have seen the crowning edifice in the address of Mr. Hutchinson. The interest I feel in this matter, and which I have no doubt Mr. Jonathan Hutchinson feels, is not on account of the wide-spread prevalence of the disease, or of any of its social relations, but from its great pathological importance. There is not a disease like it in our nosology. It is for this reason that Mr. Hutchinson has taken so much interest in the subject, and also his great master, John Hunter. In all our pathological researches, we are endeavouring to find out the why and wherefore of disease. Here is a wonderful example for us. A healthy man has a small amount of virus introduced into him, perhaps an infinitesimal amount, and the whole of his nature is changed; he breaks out into a rash over the whole surface of his body, and all his viscera are affected. For years and years afterwards any morbid process that may take place is entirely altered in its character. The man brings into the world a number of puny, ill-formed children, and the result is, perhaps, seen even in the next generation. There is no example like it in pathology. It is a large experiment made for us, and that is why so many of us take an interest in this affection.

I quite agree with Mr. Hutchinson as to this disease being a counterpart of the other febrile diseases. I have always regarded syphilis as a disease *per se* attended by its sequelæ. The physician looks upon it as a constitutional disease, and to him the words primary, secondary, tertiary, and quaternary have no meaning. Either a man has syphilis or he has not; he either has a poison in him producing all these peculiar morbid products, or he has not; and I believe that all these visceral changes that we observe are due to the true syphilitic process, and take place, I have no doubt, at the same period of time. When we examine the body of a man who has had syphilis we may find, no doubt, hard fibroid nodules in his body; but one knows, as a fact, that these nodules may have been felt in the liver, for years and years, whilst he was in a state of comparative health. Therefore I have always regarded them as a sort of *débris* of the true syphilitic deposit. That they may undergo further change, or grow, as Sir James Paget has said, I

feel little doubt, and also because we see similar inert masses increase in size in the body. But, of course, there must be some morbid process previous to their formation. We have evidence of it in those very cases where fibroid deposits have been found; we know that the patient during the exanthematous eruption really had enlargement of the liver, with affection of other organs; therefore we are bound to believe that there was a stage previous to that of the nodules, and we want to know exactly what the condition of his organs was at that period. We require more information on the subject, and we want the members of the Society to bring us specimens, if they can, of these earlier changes that take place in the viscera. That they are very marked there can be no doubt. I saw a case of this kind the other day. The patient had enlarged testes and an enlarged liver; there was dulness at the upper part of the lung, and, besides that, there was a node on the tibia and a node on the forehead. Under treatment the liver went up, the testes got small, and the lungs returned to a healthy condition; therefore there can be no doubt that similar processes in all these organs must have been going on at the same time. I have had the opportunity of examining the liver in syphilitic children, and have found what was apparently a healthy-looking liver somewhat enlarged, but very hard, and on examining it by the microscope it contained merely a little fibro-cellular tissue with a healthy structure remaining; therefore there could be no doubt that the former was absorbable material. Whether the same occurs in the adult I do not know. One specimen was brought to the Society lately. The man had the most extreme form of syphilis that I have ever seen. The number of gummata in the body was extraordinary—in the chest, the head, and elsewhere. He had a very enlarged liver, but the deposit was very like miliary tubercle, and very much resembled the specimen that was brought here by Dr. Weber a year or two ago. The possibility of meeting with these early conditions is so exceptional that we really are much in want of information as to the condition of the organs while the syphilitic poison remains in the body. Then, again, that some of the affections of the organs are acute one knows from observation. I have seen three or four cases of jaundice occur in constitutional syphilis, besides a case of acute atrophy of the liver, and I have read of one or two more cases of the same kind. The old impression was that the deposits I have spoken of were subsequent to the secondary

ones that are seen on the body, such as the rashes and sore throat. For my part I think they are cotemporary; that they are an evidence of the true syphilitic poison working in the body, and yet I must admit that they are not frequently seen. It is rather the exception to meet with those internal affections in an ordinary case of syphilis. My impression is (and to this I would call Mr. Hutchinson's special attention) that they stand in something like an inverse ratio to what are called the ordinary syphilitic symptoms. I might illustrate what I mean by the case of scarlatina, because I quite agree with him that syphilis is a true fever; having its incubation and its course, like other specific diseases. The old idea was that kidney-disease was a consequence or a sequel of scarlet fever, but there is now another opinion—and that I hold—that the scarlet fever poison is manifested on the skin, on the throat, and on the kidney; but in exceptional cases the force of the poison may be directed to the throat, when we have a scarlatina anginosa; or to the kidneys, when we have nephritis. I do not, therefore, look at nephritis as a consequence, but as a part of the scarlatina disease. I think the same of syphilis, that where it does not exhibit itself outwardly the internal organs are more likely to be attacked, and I hold the opinion for this reason. When I was commencing to work at this subject, nearly twenty years ago, my seniors, being very sceptical with regard to these syphilitic deposits, asked me for proof, and to my great annoyance the proof was found wanting. They used to look at the dead body for a scar in the groin, and then it was afterwards found that a scar in the groin did not mean syphilis. In going into the history of the case, I found that the ordinary symptoms of syphilis were wanting, much to my annoyance. Then I came to the opinion that in these cases they were really very often absent, and that opinion has strengthened with me ever since; and I may ask physicians here, or those who see cases of internal syphilis, if it is not true that they find a great difficulty in getting a distinct history of what are called ordinary secondary symptoms. The patient comes to you with epilepsy, or an enlarged liver; it is clearly syphilis. He will own to the disease, and will tell you about the sore. Perhaps he has a bad throat, or a little rash, but the whole history of secondaries is doubtful in the extreme, and yet in such a case we find internal disease. The opinion has therefore strengthened with me that there is a kind of inverse ratio between the ordinary symptoms of

syphilis, those that are generally described by surgeons, and these internal affections; and I think that may account for the different opinions that are held by many. Some say that they are due to the same syphilitic process which is going on in the body during the so-called secondary or constitutional stage, whilst others say that they are altogether subsequent to it. My opinion is that they are truly syphilitic, and yet they occur in exceptional cases. It is especially to direct Mr. Jonathan Hutchinson's attention to this that I have risen. I have no doubt that his attention has been already drawn to the circumstance I allude to, and I shall want a very confident opinion from him one way or the other about it. I will mention another reason for believing that the internal affections arise in the early period of syphilis, although their occurrence is exceptional. I have said that in these cases of internal visceral disease the testis is often affected, and just as sarcocele becomes smaller under treatment, so is absorption going on in the other organs, so that you may take the condition of the testis as a kind of gauge of the curative process. Now, if these internal affections, combined with that of the testis, are very common in the ordinary exanthematous stage, the latter ought to be known to surgeons. I am not aware what they may say now, but when I was more interested in the subject, I asked all my friends the syphilists, those who had large experience in the matter, "What do you know about orchitis in syphilis?" Many of them said, "I know nothing about it; you mean gonorrhœal orchitis?" "No," I said, "I mean syphilitic." "I do not know anything about it," was the answer I received from the most experienced surgeons. I think it is very clear from this that if the sarcocele does not ordinarily occur in the exanthematous stage, neither do the other visceral inflammations in all probability take place, and yet as both the visceral affections are sometimes met with during the early period of the disease, I consider that when they are present they take the place of the more manifest external ones.

I am very glad to hear that Mr. Hutchinson speaks of the pustular rash as occurring during the virulence of the disease. We very much want to know if we can measure the progress of the disease by the rashes. The old idea was that we had exanthems, papular and scaly rashes, at the early period, and afterwards the moist rashes; and I suppose this is more or less true. That we may get pustular rashes during the height of the disorder, I have

no doubt—during the period of the gummata. Mr. Hutchinson also speaks of this gummatous matter never suppurating. With that I entirely agree. I suppose every surgeon knows that a node on the forehead may be met with, which will soften, and fluctuate; it feels like matter, but it is all absorbed under treatment, or if it be opened it will be found that the matter is not purulent. I had a case the other day which bore upon that; it was an exceptional case proving the rule. A man in the hospital had what I thought was an abscess in the liver; that is, there was a large collection of fluid in the organ with marked fluctuation, and yet I had no doubt that the man had syphilis: then the question arose whether the syphilitic matter had suppurated. I said I had never seen such a case, although this seemed like one. When we opened it the fluid was not purulent, but broken-up syphilitic matter. It was an exceptional case, which tended to prove the rule that this syphilitic matter disintegrates, but does not tend to suppuration. I suppose Mr. Hutchinson holds the idea that this disease may run on for many years, that it may be arrested, but still it pursues a definite course. I should like to hear a little more about that, and whether we can tell exactly at what stage the disease has arrived. I apprehend that it may go on to a certain point and then stop, and that stage may last, as Sir James Paget has said, over ten, fifteen, or twenty years. There used to be a notion that a man might have syphilis, and then have a relapse; go backwards, again forwards, and so on. Now, if it be a distinct disease running its course, this cannot be. It may be arrested in the middle of its course for some reason or other; Mr. Hutchinson suggests by treatment—and perhaps this may be true; and then it breaks out afresh. We want to know, then, if by the character of the rash we can say exactly at what stage the disease had arrived. The old notion was that we had in the early period dry rashes, or scaly rashes, and afterwards pustular ones; but those who write on skin-diseases, especially the French, speak of scaly rashes at the very later periods. Perhaps when they say scaly they only allude to the psoriasis palmaris, which one sees at some of the later stages of syphilis. When I was on the Government Syphilitic Commission I questioned many of the experienced surgeons as to points of this kind, and I did not get any information from them. We heard that the disease might last fifteen or twenty years, when the man was supposed to be well, and then it would break out again. I

said, "Do you mean that the patient who has had rupia or ecthyma will come to you years afterwards with a scaly rash?" They did not know. We want more information as to the defined character of the stages, however prolonged they may be.

There is one point which Mr. Hutchinson has not gone into—whether an adult may take the disease at any of these stages, in the same way as a child who has inherited syphilis never has the primary disease, but has it at the second stage. There is a notion of this kind abroad—I believe among the French; and one distinguished man speaks of two forms of syphilis, or as acquired from a primary or secondary sore. It is an important question to surgeons in reference to contagion, whether they may be so or not. At one period it was believed that a sore in a man could only be obtained from a sore in a woman. Now we know that secondary disease is contagious; but if it be so, it has been thought that poison from the secondary disease is different from the other, or rather that, in the person who is infected, it may begin at the second period. I do not suppose that Mr. Hutchinson holds that opinion. I have no doubt myself that the disease is propagated mostly by women who simply have the secondary disease, and the man gets what may be called a primary sore. If this be true, there is an end of the question and the syphilitic poison is the same at whatever stage of its manifestation on the body it is propagated; but this is not a universal opinion held on that last condition. Only four years ago, a gentleman said here that it was perfectly useless to examine a woman under the present Act of Parliament, because the disease was propagated to an enormous extent in London, where the women were said to be healthy, clearly showing to him that there could be small ulcers existing in the genital organs without their being discovered. We have no reason, however, to suppose this because we know that secondary disease can be propagated.

Mr. Hutchinson made a remark, to which, perhaps, I have no business to allude, that is, about primary chancre having a secondary deposit in it. The subject interested me as I have seen one or two cases of it, and I rather thought that Mr. Lee's account of it explained the matter somewhat better, because it has its analogies in smallpox. I maintain that when a man has indurated chancre he has not syphilis. When Ricord first propounded that it was of no use to remove a chancre, because the poison was already working in the system, and that induration was the first secondary

symptom; I accepted his statement in a certain sense as true, but if you regard the disease as a fever and as an exanthem, it is not true, for the indurating process is merely the period of incubation: as in the analogous case of inoculation by smallpox. If I were to show any one here a patient with a smallpox pustule upon him, he would say the man has smallpox. It does not follow. If he caught smallpox in the ordinary way, and then it broke out in pustules, it would be so; but if I inoculated a person with smallpox the vesicle would form and grow for a week: during which time the patient would not be ill, although he would have a pustule; and so it would be until the end of the period, when the incubation was over; then he would break out in a general eruption, and fresh vesicles might occur in the very spot of inoculation. That used to happen in old times, when inoculation took place, and I apprehend it is much the same in syphilis. When a person is inoculated with the virus of syphilis, a chancre is formed; there is induration, but I do not see that that can be called the first secondary symptom; a month must elapse, when the whole system is infected and the eruption breaks out. I think the two diseases are perfectly analogous in this respect, and so a secondary deposit may occur in the seat where the original virus was implanted.

I have a note in reference to the course of the disease in relation to those remarkable experiments of Professor Boeck, which I do not think have received the attention they deserved in this country. Professor Boeck maintained that syphilis was a specific disease, that it was arrested in its course by treatment, but could not be cured until it had come to its natural end, and if it did not end you must make it end by putting in fresh virus; so he kept inoculating the patient until the disease was complete. That was not approved of here; in fact we thought little of it, because we said he did not inoculate syphilis, but merely took the matter from the soft sore, as he did not distinguish between the two. Professor Boeck said this question was unimportant and afterwards declared that tartar emetic might do as well. It seemed so; for he produced a number of pustules on the surface, and if you can believe in the genuineness of his cases, several hundreds of which he published, the patients did get well. It seemed that the poison was in some way eliminated by his process and could not be affected any more, just as skin-diseases will cease in a long affected part, but spread on a new surface.

I will now say a word or two in reference to the question of sero-

fula. I am rather inclined to echo what Mr. Hutchinson, rather than what Sir James Paget said on this matter. I believe I can tell a child who has had syphilis from one who has had scrofula. The appearance to me is very different. If there be a difficulty, and we apply treatment, the difference is soon seen. I can recall cases of so-called scrofula, occurring many years ago, that we did not cure, and which now we readily relieve because we give them another name, believing they are due to a different disease. I remember that, when Mr. Hutchinson brought out his work on hereditary syphilis, there was a little boy in Guy's Hospital who had a large ulcer in his throat, which was at first thought to be cancer, and then to be scrofula. He had port wine and quinine and cod-liver oil ordered him, and he was in the hospital for months. Having seen Mr. Hutchinson's book, I said, "The boy has syphilis." There were the marked teeth, nose, and head, the exact counterpart of that which Mr. Hutchinson had described. We gave the iodide, and rubbed in a little mercurial ointment under the arm, and we could almost see the ulcer heal. In a fortnight it was nearly well. As regards treatment, therefore, the two things are thoroughly different. Then the general configuration of the person is different. We had to discuss this question at the venereal commission. We had some first-rate dentists there who were sceptical with regard to Mr. Hutchinson's view about teeth, and said they were nothing more than scrofulous teeth. I think I know what scrofulous teeth are, and I know a scrofulous head and a scrofulous jaw; the upper jaw is narrow and the teeth are all crowded together. In many of these syphilitic children you see that the teeth would have been absolutely perfect, if they had not been affected in this way, but they have not the appearance or character of scrofula. Thus in every particular I am rather in accord with Mr. Hutchinson, that the two forms of cachexia are absolutely distinct.

Dr. Fagge.— It has appeared to me that there is one point of view from which the subject of the relation between syphilis and its more remote effects may be regarded, which Mr. Hutchinson has scarcely approached, and which may have important bearings. I refer to the influence of syphilis in producing lardaceous changes. I think that in the *post-mortem* room, if we find any of the viscera lardaceous, we may be sure it must have been produced by one of two causes: either the patient has had, for some time,

chronic suppuration, or he has suffered from syphilis.[1] If, in a case in which the organs are lardaceous, we look over the body, and fail to find any source of suppuration, I believe that we may be certain that the patient has had syphilis. I know it was at one time supposed that syphilis produced lardaceous disease only indirectly, and through the agency of suppuration; but experience shows that such is not the case. We find lardaceous disease in syphilitic patients when there is no reason to suppose that there has been suppuration. I do not know how this effect is produced, but I think we may say that it is difficult to conceive how advancing science can hereafter show any direct community of operation between these two distinct causes of lardaceous disease, and that we are almost justified in saying that it can only be by some general influence upon the health. It is true that lardaceous affections are the result of a change in the chemical constituents of the tissues, and so differ altogether from affections in which the pathological process is of the nature of a new growth; but if we find that syphilis is capable of inducing such a change, surely we are justified in believing that it may affect the general health, depressing it, and rendering it more susceptible to other influences which give rise to disease. It is from the relation of syphilis to lardaceous affections that I should be disposed to approach the relation of syphilis to tubercle. I know, of course, that Dr. Wilks and others believe that syphilis may produce in the lungs changes which are quite easily recognisable by auscultation, and which disappear when the patient gets better of the syphilis. There may be diffused changes in the lungs, of a syphilitic character, which get well. I cannot say that I have been convinced that that is really the case. I have seen many syphilitic patients die of phthisis. It never appeared to me that any considerable change in the lungs was really syphilitic. Even with regard to small nodules the lungs mistakes may be made. Last year a man died with gummata in the internal organs. There were some masses in the lungs, which I at first thought to be gummata. But afterwards, when the specimen had been hardened, I found that the whole of these masses really consisted of patches of caseous pneumonia: in every part of them the air-cells are still plainly visible, being filled with ordinary inflammatory products. So I

[1] Detailed evidence in support of this proposition was brought forward by Dr. Fagge at a subsequent meeting of the Society, and will be found at p. 324 of the present volume.

should not admit as gummata in the lungs any masses, the nature of which had not been carefully tested by microscopic examination. It seems to me that the fatal lung disease of syphilitic patients is not a definitely syphilitic affection of the lung itself, but an ordinary phthisis due to a remote influence upon the general health, comparable with that which causes lardaceous changes. I have formed this opinion in spite of myself. Having begun to study syphilis with regard to its effects on the skin, I have always held strongly an opposite view with regard to syphilitic eruptions, that to the last they are specific, and distinct, and capable of being recognised as different from other cutaneous affections. For instance, I have seen a patient who had from childhood been affected with ordinary psoriasis, and who came under treatment for syphilis; and, at the end of the case, after the administration of mercury and iodide of potassium, it has been easy to recognise the original patches of psoriasis, which were entirely uninfluenced either by the disease or by the treatment. So I am not disposed, generally, to mix up syphilitic affections with non-syphilitic affections; yet I cannot help feeling that, if syphilis can produce lardaceous changes, so it may produce phthisis, and, possibly, other diseases likewise.

Mr. Berkeley Hill.—I venture to solicit permission to occupy the time of the Society for a few minutes with some remarks upon some of the conclusions set forth in Mr. Hutchinson's introduction to our debate. Many of the comments I should desire to put forward have been far better stated in the eloquent and exhausting address of Sir James Paget. But let me express my admiration at the manner in which Mr. Hutchinson has performed his task. The description of syphilis which we listened to the other evening is probably the most complete that has ever been compressed into fifty minutes' reading. His digest, reducing to what order is already known in most departments of the natural history of syphilis, will form the framework to which future investigators will add the yet undiscovered details. To have put forth so authoritative and clear a summary of what is known of syphilis, reflects great credit upon our Society. At present it is rather by examining the conclusions Mr. Hutchinson has drawn that we may most usefully aid the object in view.

In the first place I would beg to demur, so far as I understand Mr. Hutchinson, to supposing that "*dualism is dead*," and that the opinion generally prevalent holds the soft chancre to be a conse-

quence of the inflammatory products of syphilis. So far from this being the case, several recent text-books on general surgery or on venereal diseases state that the soft sore is distinct in nature and origin from syphilis.

Without occupying your time with all the names I might quote, I may mention, in English, Erichsen's 'Science and Art of Surgery,' and Bumstead's 'Venereal Diseases;' in French, Fournier's exhaustive article, "Le Chancre," in Jaccoud's 'New Dictionary of Medicine and Surgery;' in German, Zeissl's 'Syphilis,' and v. Baumler's article "Syphilis" in Ziemssen's Cyclopedia. At present it would, I think, be more correct to say that while much diversity of opinion still exists, the doctrine first suggested by Ricord and established by his pupils that soft sore and syphilis are distinct in origin and nature, is gradually spreading throughout the profession. This at least is the view taken by Billroth in his 'Surgical Pathology.'

A more important matter is the view that syphilis is a *specific* fever, to be classified on that ground with smallpox or scarlet fever. This seems to me misleading, and likely to produce mischief in the prevention and treatment of the disease. Mr. Hutchinson tells us that better acquaintance with the acute eruptive fevers shows that they vary quite as much as does syphilis in length of stage, in degree of severity, and in the occasional omission of some of their phenomena. Let them vary as they may, any one of the recognised specific fevers taken as a whole is, I still think, strikingly different from not only the ordinary course of syphilis, but from any extraordinary course of that disease.

By taking advantage of these irregularities in the course of acute fevers, by abruptly limiting the duration of the secondary period, and by pushing into the back ground the later phenomena as merely local affections, Mr. Hutchinson produces a picture that at first sight bears a resemblance to the eruptive fevers. This resemblance will nevertheless not bear close scrutiny. If our discussion establishes syphilis among the eruptive fevers, it must I think be by the contribution of new facts not yet laid before us.

An important point with Mr. Hutchinson is to draw a great distinction between the so-called secondary and tertiary symptoms of syphilis, that the former may be considered effects of general blood-poisoning, and the latter only local remanets of the exhausted infection.

It is unnecessary to encumber the discussion with additional

examples of the acknowledged characters of syphilis, already so well drawn by Mr. Hutchinson, for a different appreciation of the facts he enumerates form an ample structure to suffice to establish and support the theory which sets no absolute or definable limit between the several periods of the disease—the primary, the secondary, the latent and tertiary stages.

I contend that at no time can a lesion of syphilis be termed a mere local affection, still less that the blood is ever free from participation in the disease. On the contrary, from the very first appearance of induration at the point of contagion to the latest syphilitic phenomenon, we have to deal with an essence that influences all the tissues.

Certain characters Mr. Hutchinson has grouped together as common to all periods of syphilis, to that which he would have us call the season of local affection, the so-called tertiary period, as well as to the earlier stages when the blood and all other tissues are affected.

These characters I shall claim as sufficient evidence of the essentially chronic course of syphilis that renders it distinct from the recognised fevers. Mr. Hutchinson selects others on which he relies to prove that a radical change takes place in the nature of the disease when the symptoms become apparently isolated rather than general, and follow each other at long rather than at short intervals.

He tells us that *symmetry* is a characteristic while the blood is infected; both sides of the body are attacked and several of the regions which can be searched by the eye are simultaneously the seat of morbid change.

Acknowledging that little is yet known concerning the affections of the viscera and deeper structures at this early period, Mr. Hutchinson suggests that they usually occur simultaneously with the early eruption on the skin, but spontaneously subside after a brief existence; in this respect they would correspond to the ephemeral symptoms of the surface. It may be that there is some truth in this conjecture, for it is no more than conjecture; nevertheless, I would remind him that recent contributions to our knowledge of early visceral syphilis indicate these affections to be similar or identical with the later so-called tertiary affections with which we have been long acquainted. But be this as it may, symmetry, Mr. Hutchinson confesses, is a distinction of abundance rather than of character; and if he recollects how gradually the

general widespread rashes that first appear are replaced by succeeding rashes each more limited than its predecessor, we may be pardoned for not seeing in the cessation of symmetry a very clear demarcation between the early and later periods of syphilis.

Another distinction is the spontaneous resolution of new growths that is constantly witnessed in the early stages. Though very usual then, he admits it is not peculiar to the secondary period, for a little further on in his description of tertiary products, Mr. Hutchinson remarks that their spontaneous resolution often occurs.

Indeed, I think he will acknowledge that the natural tendency of every syphilitic morbid process is to cease without permanent loss of tissue; for, though ulceration usually attends the growth and waning of tertiary affections, such a complication is occasionally the accompaniment of early syphilides.

In addition to symmetry and spontaneous resolution there is the cessation of the contagious condition which Mr. Hutchinson believes to happen when the general rashes disappear and the latent period arrives. I shall endeavour to show that termination of the contagious condition is also gradual, and not a phenomenon suddenly produced in any case.

The facts which lead me to hold syphilis to be a chronic malady, different in nature from a recognised fever, are:

1. The histological identity of the morbid processes at all periods.

2. The frequent appearance soon after contagion of those affections which are commonly delayed to a late period.

This precocious development of gummata shows the identity of the disease in the early and later stages, and probably also the intensity of the virus, for early gummata and other tertiary affections are most frequently observed in severe cases of inherited syphilis where the parent is recently infected. Some of the most typical of visceral gummata are seen in stillborn children.

3. Certain antidotes can subdue every process of syphilis even to preventing contagion, and these antidotes are effective at all periods, both early and late, of the disease.

4. But the most important evidence for my purpose is the long duration and gradual extinction of contagion, in a manner peculiar to syphilis and quite distinct from the contagion of acute eruptive fevers.

Syphilitic contagion is not volatile nor does it appear to be soluble in fluids, but to require an organized cell for its conveyance, the blood-corpuscle, the spermatic cell, and the ovum, being those against

which the best evidence of guilt in this respect has been collected. Propagation takes place in two ways, by contact and by hereditary transmission.

The former, or propagation by contact, is most frequent during the secondary period, but ceases in most cases when the latent period is reached, that is, in two years or thereabouts after infection. But it is important to note that it does not cease abruptly, as should be the case if syphilis were analogous to the acute eruptive fevers, but may reappear at intervals during relapses of the disease before it finally dies out.

The second mode, propagation by inheritance, very active during the early stages of the disease, retains its power through the latent and tertiary periods, requiring in certain individuals many years to pass away before it becomes thoroughly inert.

Again, a very distinctive point, during this period the virus is not changed in nature, but only in potency by lapse of time. This peculiarity is shown in a striking fashion. Syphilis, whatever phase it presents in the parent, always developes in the infant as early general disease. Tertiary affections, indeed, notin frequently develope in the infant with the earliest cutaneous rashes, when the parent has not been long infected.

Yet another point which demonstrates also the continuity of the disease, namely, it is immaterial whether a syphilitic infant have been infected by a secondarily or a tertiarily affected parent; the secretions of such a child communicate the ordinary form of syphilis to his attendants as readily as do those of an adult in whom the early exanthems and erosions are in full career. These characters of the contagion are insuperable to any allocation of syphilis with acute fevers.

This curiously slow exhaustion of contagion has been fully described and put beyond question by Dr. Kassowitz, whose remarkable memoir has recently been noticed in the 'British Medical Journal.' When the disease of the parent is not treated, or only insufficiently so, its course is commonly thus. While infection is recent the first fœtus is born dead prematurely, the succeeding fœtus may also be prematurely born, or if born at term dies in a few hours. The next child is attacked soon after birth and succumbs in a few weeks. When years have followed the parent's infection the child, apparently healthy at birth, shows at the end of the fourth week symptoms of the disorder. In

succeeding children the disease may delay its appearance as late as to the end of the third month, but is never postponed beyond that time. Kassowitz has demonstrated that three years must be expected to elapse after infection before a viable child can be born, and many more years before the influence is finally exhausted. When the parent is subjected to careful mercurial treatment a healthy child may be born long before the disease is finally quelled.

The exhaustion of the contagious influence is so regular and gradual that the fate of the fœtus can be foretold when the age of the infection in the parent is known, and *vice versâ* the duration of disease of the parent can be calculated by the symptoms of disease in the infant.

Here I should like to suggest the possibility of a similar effect in acquired syphilis to this enfeeblement in the hereditary disease, but for which I have insufficient evidence to propose to establish as a character of syphilis. It is this—sometimes the course of the disease in a patient is mild when he has contracted it from a person whose disease is of long standing and approaching the latent period.

On the other hand, in cases of severe course and copious development the patient has been infected from a person with recent syphilis. I have only records of thirteen instances where I was able to compare the infected patient with his source and in whom I had sufficient reason to believe that the supposed source was the real one. Moreover, I believe that conditions existing in the individual himself are powerful over the course the disease shall take. Hence, while bringing forward this fact as a suggestion I refrain from urging it as a law, until ample evidence has been collected.

Dr. Wilks has told us that he long ago suggested that those who suffer from the inner and deeper forms of syphilis have a poor story to tell of the superficial affections of the early stage. This, which is doubtless quite correct, I probably learned from Dr. Wilks, and forgot that I had done so, for I have been in the habit of supposing that I discovered this point myself. I rejoice to be reminded that the existence of this character rests on Dr. Wilks's testimony.

Somewhat the converse has occurred to me as being also the fact; namely, if a man have long-continued palmar or plantar psoriasis, for example, we may pretty confidently assure him that he will not be troubled by the deeper affections of syphilis; that if the disease expend itself mainly and continuously on the skin, he will probably escape the visceral lesions which are so fatal.

The characters which I have recited are, it seems to me, entirely

different from those of the acute fevers. These maladies do not commence with an initial lesion; they have a rapid course abruptly terminated by convalescence, free from relapses, and their volatile contagion does not remain smouldering in the parent to break forth into flame year after year in the child. Lastly, no chemical agent is their antidote.

Most unlike such disorders, syphilis consists of a series of morbid processes all essentially identical and almost repetitions of each other. Proceeding from the imbibition of a virus, copious at first and besetting all the tissues, these processes become fewer as the energy of the poison diminishes, and ultimately disappear altogether unless roused again and again into activity by favourable condition of the poisoned individual.

There is another point in which I desire to say a word. Mr. Hutchinson did not allude to the conditions of the individual which prolong the course or increase the severity of the disease. I mean, for example, the lymphatic temperament or the scrofulous diathesis, or those artificial states of debility produced by alcoholic indulgence, great bodily fatigue, exposure, or insufficient nutriment. Such are commonly held to be powerful factors in determining the severity and possibly the duration of the disease. At the least they must be allowed for when considering what influences determine the prolongation of syphilis into the tertiary period.

With regard to scofula and tuberculosis Mr. Hutchinson has pointed out that syphilis never merges into them nor have they any ancestry in syphilis. Doubtless much that was once considered scrofula is really syphilitic, and when all that is tubercular and that which is syphilitic is separated from the truly scrofular, there is so little left that I have been inclined to wonder if there is such a diathesis. It will be interesting to hear from Mr. Hutchinson how he distinguishes the three diatheses.

Mr. de Meric.—I may be permitted to allude to Mr. Hutchinson's address as one worthy of himself; one which has not disappointed any of us. We know well that anything emanating from him must be eminently philosophical and true. But I am afraid that, like all men of great merit, and men thinking a great deal upon one subject, he may have been carried away by trying to throw an enormous deal of unity, uniformity, and order into his views with respect to syphilis. The fact is, such men as Rouse and Brown in Scotland

aim at simplification. Brown said it was a sthenic disease, or that it was an asthenic disease. Rouse said that it was inflammatory or non-inflammatory, and so on. So you find Mr. Hutchinson addressing you in this way, "with the desire"—I use his own words—"to simplify and make more orderly one general view of the subject." Now, in simplifying the subject, we must be very careful to adhere exactly to the pathological facts that lie before us, especially in practice; and, in the investigation of the disease, uniformity may, perhaps, degenerate into a little confusion, and we know that the way in which certain diseases have been divided for us lightens our task in practice. We follow those who have examined and studied the disease and divided it, and we follow it in our practice as far as we can. Hence, *in limine*, I would say it is rather a heavy task to attempt to simplify this subject to any very great extent. But, rather than take a general view of syphilis, rather than express any opinion of my own, I will pay due respect to the address itself, and endeavour to review it. I find that it may be divided into a certain number of heads: first, one virus; secondly, syphilis is a special disease or a special fever; thirdly, there is such a thing as cell-growth in syphilis which may be followed by phagedæna; fourthly, syphilis presents analogous features at different stages; fifthly, tertiary symptoms are local sequelæ; sixthly, certain contrasts exist between inherited and acquired syphilis; and lastly, syphilis is not convertible into struma. I shall not take the liberty of alluding largely to every one of these points; I will only hint at a few of them, and endeavour not to trespass too long on your patience. First, as to the one virus. Here our distinguished friend has gone a step backwards; he seems to have wished to revive the opinion of Hunter, Ricord, and others respecting that one virus; and in this respect he has had a very great supporter in Sir James Paget, who also concurs with the opinion of Ricord, that there is but one virus, but that it will act differently according to the individual upon whom it works. That was Ricord's view; but he was too busy in separating the virus of syphilis from that of gonorrhœa to pay much attention to what might have been said with respect to the virus itself. He separated, as Hunter had confounded, the two. Several other opinions have been formed, in order to get rid of certain difficulties in the way. I see that Mr. Hutchinson has taken refuge respecting the differences in the virus in germs. He says, in one portion of his paper, that, if a syphilitic secretion contain the germs

of the actual disease, it will convey it; but, if these germs be absent, it will not. In another part of the paper, he accounts for the soft sore in this way: a truly syphilitic ulceration may become phagedænic, and pus, which is in some degree spoiled, may be the origin of the soft sore. There are various ways of accounting for that soft sore. Why take refuge in those germs, which, I suppose, Mr. Hutchinson has no more seen than either Pasteur or Lister? At all events, there was that supposition of my friend Clerc in Paris, that a soft sore was simply the descendant of one that had been inoculated, and that the sore inoculated upon an individual already having syphilis produced a kind of hybrid afterwards. This was a supposition and a theory. But, after all, why quarrel with the dualists? There are very few books teeming with as much clinical and pathological truth as that of Bassereau, which has stood the test of twenty-four years. The book was published in 1852. I will no further allude to it than to say that it did put upon pretty safe ground the view that the soft sore had existed at all times, and had been confounded; and that eventually, at the pestilence of 1694, the soft sore really made its appearance. It would be out of place, however, to discuss the matter here. Then we come to the question of the specific fever. It is no wonder at all that Mr. Hutchinson should have established his analogy. I am glad to find that he is so true to his colours; because, several years ago, in another place, I had the pleasure of being present when he propounded that view. He certainly might well do so, because, in a physiological point of view, the resemblance is very great. There is only one of these specific fevers, however, where the analogy is quite correct—that is variola—because there we have an original sore; the difference being, however, that variola can be caught by a tainted atmosphere, whereas the other cannot; and, besides that, there is, which is a little inimical to the analogy, the hereditary transmission afterwards. It is plain that there are certain analogies in disease; but why should we force them. Why should we not take one as it is, and the other also? Analogies are very useful in certain respects, but not in all. Then there is a subject which Mr. Hutchinson has investigated with great talent. He has shown us well, as has been stated by Dr. Wilks, that there is a peculiar growth, a peculiar deposit of masses which takes place in syphilis, and which is peculiar to that complaint. We see it at first with the hard chancre, we see it afterwards with an ulcer; we see it in the nose, in the gummata, and so on.

Mr. Hutchinson says in his paper, "I am not speaking of inflammatory processes, but of cell-growths;" and that we all acknowledge as the peculiarity of the complaint. But here the author has run a little too far respecting the manner in which these masses may be destroyed, and he has indulged in a very lugubrious picture of phagedæna. I can quite understand that, by the power of the organism, such growths should disappear and become absorbed; in fact, we are all aware of that most astonishing thing, how the very hard mass surrounding a hard chancre will, after a certain time, and by treatment, entirely disappear; but we should recollect, also, that another process may take place: it may tumble down; an inflammatory process may take place, and then phagedæna may occur. Mr. Hutchinson has ventured upon an expression which certainly was very bold, that syphilis is the originator of all phagedæna. This is going somewhat far. I must say that I have seen, in the course of twenty-five years, a great many very bad cases, both in hospital and private practice, and phagedæna has been rather the exception than the rule. I do not allude to those unfortunate beings in hospitals in whom neglect, poverty, and want of cleanliness, bring on the most destructive processes; but I do not see why we should put at the door of syphilis, which has enough to answer for, all the mischief which may be brought on by phagedæna, to such an extent as to say that when the wizard of syphilis evokes the monster of phagedæna the part is lost. I must say that it often happens that such results are by care avoided. Mr. Hutchinson has been particularly lucky in his analogies respecting the different features which we see in the stages of syphilis. I must adhere, however, to what I look upon as stages, rather than put the whole disease into one lump, as he wishes. He has pointed out that there are different parts which are gradually affected, as the skin, mucous membrane, and so on; and he has shown his analogies wonderfully; but, having in some degree ignored the tertiary stage, he has to some extent overlooked what he himself stated; and that is that, in the secondary stage, there is the peculiarity that masses that are deposited will become absorbed, whereas, in the third stage, these masses are most likely to run into very destructive inflammation; therefore, when he speaks of the great analogy existing between the secondary and tertiary stages, he very wisely also points out that there are those differences; and it is exactly those differences, as they affect practice, that we principally look for in this paper. Now we come to the sequelæ—the tertiary

symptoms; and this is a question which has been treated in his usual masterly manner by Sir James Paget; that is, the question whether we have on one side a blood-disease and not on the other. It appears to me that Mr. Hutchinson has relied a little too much on symmetry; in fact, if he have any failing, that is his failing—he wishes to bring everything into mathematical order. This symmetry respecting syphilitic phenomena, to my mind, does not exist. Recollect what we see every day. First, we see a chancre that is uniform, at all events. Then we have rashes; recollect the roseola—recollect the little psoriasis, the little impetigo scattered all over the body; then we have a sort of glandular enlargement, which may be very unsymmetrical. In fact, the symmetry which seems to be the basis of his theory does not seem, to those who examine the question without any preconceived notions, to be of much importance. Then comes the want of symmetry in the tertiary symptoms. As it is said by Sir James Paget very properly, the disease is on its decline; it no longer has a firm hold on the patient, and there are only some points here and there which show a certain amount of disposition to take on a pathological state, and here the remains of old scars have been called in to prove that there may be a renewal of the disease that had once existed. If I may be so bold as to criticise anything that has fallen from Sir James Paget, I would say that the simile respecting scars of leech-bites does not exactly tell on a lurking pathological process of a secondary kind, which might have been going on in the cellular tissue at a deeper portion, and which, under certain excitement, afterwards recurred. I am sorry that the leech-bites gave Sir James Paget any trouble, but they evidently were not of the pathological description belonging to the remains of a secondary eruption. The great contrast between acquired and inherited syphilis has been touched upon in a masterly manner. That contrast was quite congenial to Mr. Hutchinson, owing to his great labours respecting interstitial keratitis. Here he was at home; and we all agree that his labours in that respect have been of enormous use to our knowledge of the complaint. It is an extraordinary thing that children who have suffered from syphilis should remain in the state of latency so long, and then have interstitial keratitis shown afterwards. The only passage that I did not exactly understand was that which stated that such keratitis had been noticed by Mr. Hutchinson where he had been able to trace infantile syphilis, but that he had seen that same keratitis also

in people who had never had the complaint. This would seem to require some little explanation. The last point is extremely simple, and it has been touched upon by other speakers, whether syphilis is convertible into struma. It has been justly said by Mr. Hutchinson that disease itself will weaken the individual very much, and that treatment will weaken him; but it should never be forgotten that syphilis will come down upon individuals of different kinds; and if they should be already prone to certain strumous dispositions, of course it will be shown afterwards. I will allude to hydrocephalus. I have seen many children who have lived after infantile syphilis with marked symptoms of hydrocephalus. I recollect three or four boys whom I was able to follow up to the age of thirteen or fourteen, who had very big heads. Many people would at once look upon this as a strumous deposit; whereas those who were acquainted with the history of the case, knew perfectly well that it was syphilis. Before I sit down, allow me thank Mr. Hutchinson for the excellent manner in which he has introduced this discussion.

Adjourned Discussion, March 7th.

Dr. Broadbent, after some preliminary observations, expressing his assent to the doctrine of the unity of syphilitic virus, said:—Respecting the relations of syphilis and scrofula, my experience is simply confirmatory of that of all previous speakers, that there is no direct relation between syphilis and scrofula; that scrofula is not simply syphilis in the third or fourth generation. But Mr. Hutchinson invited contributions of experience of the later history of cases of congenital syphilis, giving as the result of his experience, that they were not liable to syphilitic affections of the nervous system, to gummata, or to nodes in any special degree; or to any serious form of disease later in life, and that, in point of fact, congenital syphilis after the early dangers did not render a life less valuable. As to the first of these points, my experience coincides with that of Mr. Hutchinson. I have seen a great deal of syphilitic disease of the nervous system, but very few instances indeed of diseases of the brain in the subjects of congenital syphilis, and those instances so exceptional that they would rather tend to confirm the rule. As regards the liability to gummata and to nodes, and the tertiary forms

of syphilis, I confess I should not quite have come to the same conclusion. Of course, I see these things from a different standpoint; but I have seen large gummata in the tongue, large cutaneous gummata, and more especially, and not at all infrequently, the peculiar rapidly destructive ulceration of the soft palate and pharynx, which is so characteristic of tertiary syphilis. I should, of course, put these ulcerations side by side with gummata as tertiary lesions, and I should not have been led to quite the same conclusion respecting their infrequency. But the most important fact that Mr. Hutchinson stated was, that these cases of congenital syphilis are not liable to any serious forms of disease in after-life, and that, in point of fact, they will enjoy (I suppose according to his idea) an average longevity. Now, it has struck me, and this matter has occupied my attention, that the syphilitic physiognomy, the usual signs, in fact, of congenital syphilis, are very rare in and after middle life. My own experience is that they are very seldom seen, and I should venture to say from my own experience, that somehow or other these signs of congenital syphilis do disappear before or at middle life, from some cause or other. Mr. Hutchinson stated that in the cases which he has watched from youth upward, two of the patients had died of contracted granular disease of the kidney; but this he attributed to the prolonged administration of iodide of potassium. I am in the habit of giving iodide of potassium in large doses, and over long periods; and I have only seen one case of contracted granular kidney after the administration of iodide of potassium, and in that case another known cause of the disease, namely, frequent pregnancies, had been in operation. Unless, then, Mr. Hutchinson has very strong evidence that iodide of potassium is capable of giving rise to this disease, I should myself be disposed to attribute the contracted granular disease of the kidney, of which the two patients died, to the congenital syphilis. We are all familiar with the fact that cases of contracted granular kidney, to an extreme degree, occur early in life, and before there has been time for the operation of the usual causes of this condition. It may be a matter of coincidence, but I have seen two cases in which there was this contracted granular disease of kidney early in life, with no history likely to lead to this condition in subjects of congenital syphilis. I think, perhaps, also that some of these may die off from amyloid disease. Dr. Fagge, at the last meeting of the Society, stated that the only two recognised causes of amyloid disease were syphilis and suppuration. I think

most physicians here must have seen instances of amyloid disease early in life, in which there had been no suppuration, no disease of bone; and if Dr. Fagge's dictum be true (and I will pay great personal respect to it, for it would not be lightly uttered), then we are left to the conclusion that congenital syyhilis must be a cause of amyloid disease, which will carry off some of these cases. Of four cases of amyloid disease of the liver and kidneys in children that I have seen, one was distinctly associated with congenital syphilis. I come now to the question of the relation of syphilis to the specific fevers; but here I must ask permission to circumscribe somewhat the class within which the comparison should be made. There never has been any doubt that syphilis was a blood-poison, and that therefore it must present analogies with other blood-poisons. But if we get no further than that, we shall not have gained much ground. Among these blood-poisons, however, there is a well-known class, which stands limited by certain common characters—the class of fevers. These characters are—as is very well known—first, that the poison is introduced from without, and not generated within—the poison is derived from a pre-existing case of the disease; secondly, the poison is reproduced in the individual, who therefore becomes a source of contagion; thirdly, these fevers have a very definite course and duration; and the fourth important characteristic is, that they occur only once during life. It is to the diseases comprised in this group that syphilis is, I believe, compared by Mr. Hutchinson. And this would exclude from consideration pyæmia, mentioned by Sir James Paget at the last meeting, except for the purpose for which he introduced it, as an illustration of a blood-disease, symmetrically developed in the first instance, and becoming later on unsymmetrical in its manifestations; but it excludes ague (which he also mentions) completely. Ague has nothing in common with the class of diseases of which I have spoken. If the affinity of syphilis with these diseases can be established, we do make a very important step; we place syphilis in its true relations, and we have in syphilis, with its slow and deliberate processes, an opportunity of studying, as I have already said, the invasion of a blood-poison much more carefully than in the more rapid course of fevers. There is only one of the conditions which I have mentioned, which syphilis fails to fulfil, and that is its definite course and duration. If we make the tertiary symptoms a part of the syphilitic process at all, then, I think, by scarcely any permissive latitude can we bring syphilis into the same

group with the well-defined fevers. Mr. Hutchinson limits the febrile stage of syphilis at which we have a blood-poison to the second stage; and he places the tertiaries among the sequelæ, because they fail in symmetry, they break out on the application of external violence, or on other exciting causes. The whole question is, whether the tertiary stage of syphilis can properly be considered as sequelæ. We have heard from Sir James Paget that well-characterised blood-diseases, such as pyæmia, in their later stages cease to be symmetrical; and that under these circumstances want of symmetry does not imply that the disease has ceased to be one in the blood. And there are blood-diseases, such as gout, in which this symmetry is as a rule wanting. But the tertiaries of syphilis cannot very well be compared with the sequelæ of other fevers. There is something specific about tertiaries; they come from syphilis and from nothing else. Now, we cannot say that of the sequelæ of other fevers. With regard to the instances given by Sir James Paget of thrombosis, necrosis, and the like, there is nothing specific about these. If we look through the whole range of sequelæ of acute specific fevers, we find it very difficult to produce a single parallel to a tertiary manifestation. We cannot consider the scrofulous manifestation following measles, or the purulent catarrh, as semi-specific, in the same sense as the tertiaries of syphilis. Perhaps the only illustrations we can find are the parotid bubo of typhus, which is often unilateral, and the renal affection of scarlet fever. Perhaps the recurrent glandular affections and recurrent eruptions of scarlet fever, and even the most striking examples of kidney-affection in scarlet fever, would be rejected if we took Dr. Wilks's view of the matter, and considered it merely as scarlet fever attacking the kidneys instead of the skin or in the throat. As to Mr. Hutchinson's hypothesis, that the tertiary manifestations may be re-grown in deposits or remains which were laid down during the secondary period, I am hardly prepared to give an opinion; and I would ask Mr. Hutchinson in reply to develope it further. I think that this similarity of histological product has been pushed too far, and that we must interpret histological structure by clinical history; and the clinical history of these tertiaries is so different from the clinical history of secondaries, that I think, considering the limited range of histological variation, we should not attach very much importance to the similarity of the gummata in their structure with such secondaries as we know, and with the structure in the primary chancre. And

if we make too much of this similarity, we are leaving out of sight another very important characteristic of the syphilitic process—the process of destructive ulceration. But there are other difficulties in the way of accepting that the tertiaries are the sequelæ of the syphilitic fever. There is the fact mentioned by Mr. Berkeley Hill, substantiated by my own experience, that tertiaries are much more likely to occur in cases where the secondaries have been very slight. Another objection has also been advanced; namely, that a parent who has reached the tertiary stage of syphilis may have a child who shall have syphilis, but shall begin with the secondary stage. If a disease is communicated in any other than as a mere tendency or proclivity, it can hardly be transmitted otherwise than as a blood-disease. I may here take the opportunity of asking whether infantile syphilis, or rather hereditary syphilis, is so severe and fatal because of the infancy or because of the heredity. There is still one other difficulty which I find in accepting this idea, that tertiaries are simply sequelæ; it is that of a woman may become syphilitic by contamination of a syphilitic fœtus; that under these circumstances the woman may have no secondary syphilis whatever, but go straight to the manifestation of tertiaries. My own observation leads me to the opinion that a woman may get syphilis through the fœtus, and that in these cases the syphilis runs a very different course from that which it runs when the woman is infected directly in the usual method. I have very fully set forth my difficulties in the acceptance of the idea that tertiaries are mere sequelæ. I have done so, not because I object to the theory itself which places syphilis among the fevers, but because I have great confidence in that theory, and desire to see the difficulties removed. I have, indeed, so firm a confidence in this theory, that upon it I base the opinion that we may be called upon to revise our do-nothing treatment of fevers from facts which we see in the history of syphilis.

While, again, in the ordinary sequelæ of fevers there is nothing which can be called specific, in the results of fevers there is something which is markedly specific, and that is, immunity from subsequent attack. Sir William Gull long ago showed that this immunity could not be explained on the hypothesis that the fever-process had exhausted fermentable materials, but that it must be due to some change in the tissues themselves. I think it is not unlikely that the specific immunity of fevers may be found to be represented and carried further in the tertiaries of syphilis.

Dr. Buzzard.—I propose to allude to two points in Mr. Hutchinson's address—first, as to the occurrence of visceral lesions in secondary syphilis; and second, the hypothesis that tertiary gummata are local regrowths in formations left over from the exanthem stage. With regard to the first of these points I shall confine myself to syphilitic affections of the nervous system. It must be remembered that long after various forms of nervous lesion had been referred to syphilis, it was supposed that, in every case, the mode in which the nervous system was affected was indirect, and by means of some disease of the bones of the skull. It was only in process of time that it became known that syphilis could cause directly disease of the membranes, of the interstitial neuroglia, and, still later, of the blood-vessels of the brain. Although it is now a great many years since syphilitic affections of the nervous system were described as being capable of happening during the secondary stage, the idea of the frequency at all events of such occurrence has not penetrated largely the minds of medical men, at least in England. They are still very generally supposed to be confined to the tertiary stage. Since the last meeting of the Society, I have examined my notes of a hundred cases of affections of the nervous system, which I diagnosed to be due to syphilis. The mean age of the patients was thirty-five years. I put in this list no case in which any disease of the circulatory apparatus or kidneys could be discovered by examination, and in only two instances was there any history of antecedent injury. Of these hundred cases, I was able to obtain the history of the probable date of infection in eighty-three; and, out of those eighty-three, in fifty-six the nervous affection occurred at a date longer than five years after the infection, and twenty-seven at a date under five years from the infection. Dividing the period of twenty-five years (which embraced the longest interval that was ascertained) into periods of five years, the first period—that under five years—included a third of the whole number of cases. The next period, namely, that from five to ten years, embraced twenty-three cases; and that from ten to fifteen years, twenty-five cases. Then they fell rather rapidly; from fifteen to twenty years the number was only six, and from twenty to twenty-five years only two. Under two years the number was six; under three years, four; under four years, six; and between the fourth and fifth years the number of cases was eleven. These were cases of hemiplegia, of convulsive disorder, and of paraplegia. There is a point of interest that arises here. I found no case of

optic neuritis occurring in any patient at a date less than three years from the period of infection; and out of these hundred cases, I found optic neuritis, or atrophy of the optic discs, arising from optic neuritis, in no fewer than fifteen cases. There was paralysis of muscles of the eye in twenty cases; there were appearances of disseminated choroiditis in five cases, and old iritis in two. In the period from three to five years there were four cases in which, combined of course with other symptoms, there was optic neuritis, three which presented paralysis of the eye muscles, and one disseminated choroiditis. In reference to the character of the affections in these very numerous early cases of syphilitic nervous disorders, there was nothing very remarkable. I can only speak of the symptoms they presented, because none of them died; but, judging of the pathological condition from what one could observe of clinical signs, there was no marked difference between these cases of early occurrence and those of persons who showed nervous lesions at a very much later date. There were, in a few instances, some exceptions to that. There is a peculiarity in some of these early cases of syphilitic nervous affection. The patient complains of fainting, and you see a condition of contraction of the veins where they are superficial enough to be visible. You see the pupils becoming large, and you find that the action of the heart is accelerated. These are symptoms of irritation of the vaso-motor nerves. I have only found them in cases of early syphilis. I have seen a condition of mental excitement, approaching mania, in several cases in very early (secondary) syphilis.

As regards the second point that Mr. Hutchinson mentioned, that tertiary growths represent local renovation of long-existing germs, I should say that I incline to agree with him, and also with the opinion of Dr. Wilks. It is most likely that the lines of the future gummata are laid down during the exanthematous stage. The absence of symmetry is peculiarly evident in syphilitic nervous lesions. The gummata in tertiary syphilis occur almost constantly on one side or other of the brain; and if there should, at first sight, appear an exception in the case of optic neuritis, which is almost always double, it should be remembered that optic neuritis is produced by a single tumour acting upon a single hemisphere. I have seen numerous cases in which hemiplegia or paraplegia has recurred more than once. This would suggest, not the continuance of a blood-disease, but a local deposit which every

now and then has led to renewed action. The absence of pyrexia, which is so significant a part of the second stage of syphilis, is another symptom. Did I not shrink from rushing in where Mr. Hutchinson and Dr. Wilks have feared to tread, I should suggest that we might even hazard a guess as to the particular tissue in which that local deposit occurred.

The researches of the last few years tend to show that the lymphatic system is much more widely spread than was formerly supposed. In addition to the long-recognised vessels and glands, not only the serous sacs (pleura, peritoneum, &c.), but also, amongst other structures, the peri-vascular canals, the fissures between the canaliculi of the testis, and those ill-defined pouches of loose connective tissue which allow a muscle to glide over periosteum or a subjacent muscle (I do not refer to the true bursæ mucosæ) have been discovered to be lymphatic spaces, whilst the tonsils are collections of follicles representing lymphatic gland in its simplest form. The frequent occurrence of gummata in situations of this kind is notorious. I might suggest that, during the exanthematous period of the disorder, germs, or potentialities of disease are imbibed from the tissues, are carried into the various portions of the lymphatic system, and there lie inert for a time, until some circumstance, with the nature of which we are not acquainted, calls them into action; and that then the outgrowth occurs.

Sir W. Jenner (who was requested by the President to address the Society) said: Having heard but little of this discussion, I am sure the Society will excuse my not entering fully into the subject. There are one or two points, however, to which I would refer. I must own that, although I have seen a good deal of what I would call acute specific diseases, I am not acquainted with that marked symmetry which has been alluded to. Take the eruption of typhoid fever. I know nothing symmetrical about it. I should say that want of symmetry is one of its characteristics. Ordinary psoriasis (such has I can produce in some persons at will by irritating the skin) is symmetrical; yet I do not think it is a blood-affection, but rather that it is a potentiality of developing tissue I cannot see that symmetry necessarily arises from a blood-condition; nor can I see that certain diseases in which the blood is damaged are necessarily attended with symmetry.

There is one specific disease, one acute febrile affection, to which,

without entering into the question of tertiary conditions, I would call Dr. Broadbent's attention, in which there do seem to be sequelæ of a very peculiar nature which do not appear to be mere accidental disturbances of nutrition. Diphtheria is unquestionably an acute specific disease, having its origin in a special poison. It runs its course, and then, perhaps, in a fortnight or three weeks, or a month, you see results that you would never dream of referring to the attack of diphtheria except by a series of observations and by a long process of reasoning. The child begins to see badly, and you say that the muscles of the eye are affected through the nerves; then fluids return through the nose; then the child begins to stagger. There, it seems to me, you have the sequelæ of fever, not in the sense of mere disturbance, but as a part of the residue of the disease itself; something quite different from the scarlet fever affecting the kidneys or diphtheria affecting the brain, but something special. Then I would speak of congenital syphilis. Of course we all see these internal syphilitic affections; but surgeons, perhaps, are better acquainted with them, as a rule, than physicians; congenital syphilis, however, does come under the observation of the physician, and I cannot help seeing here something that is not blood, but something in which the blood may, and probably does, share the injury. I see the father, as Dr. Broadbent has put it, the sperm, something anterior to the blood, the sperm on the one side and the germ on the other, when it is a mere cell, going on to division after division, and yet every one of those particles partaking of the syphilitic character. And then I see the first development in the tissues, in the tissues which are being formed before and in common with the blood. It appears to me that these primary cells are the real seats of the change. It seems further, as in cancer, that the father or the sperm does give to the primary cells a potentiality of development; and that seems to me to be the key to these congenital diseases; a potentiality of development which shows itself in the syphilitic child at the earliest period, but still goes on until the second dentition and afterwards. We see this potentiality of development coming by the parent to its offspring in all the tissues visible to the eye,—in the colour of the eye, the formation of the pigment, the formation of the whiskers, and so on; and this potentiality is seen not only in formation, but also in decadence. Thus the father, by the stimulus of his sperm, has given to every tissue of the child this potentiality;

even the teeth may fall out early or not, according to the power given to the germ, but not showing itself till late in life. Potentiality of development possessed by the germ if inherited from the mother, or impressed on the germ if inherited from the father, then, and not the state of the blood, not any visible organic change of structure, is the real pathology, it seems to me, of infantile, *i. e.* congenital syphilis.

Dr. Moxon: Before proceeding to any more special remarks, I should like to felicitate Sir William Gull and myself, and those who took the same view, upon the very distinguished support that we have just had in regard to our view on the pathology of cancer, which I rather understood Sir William Jenner to oppose on a former occasion, though I now understand him to say with us that it fixes itself upon kinds of tissue rather than that it belongs to the blood. That only in passing. I will now address myself to the matter before us. Mr. Hutchinson has asked specially for facts. I thought I had brought with me some facts noted down that I had collected; they were not exactly what Mr. Hutchinson asked for, such as were required to support or oppose very special lines of theory which he had spun out; they have no bearing either for or against such theories, but have only a general interest to the profession (so, at least, I thought), representing my own experience with regard to the time at which visceral syphilis is fatal. I found in the course of fifty-six inspections upon persons who had died of visceral syphilis, that the average period at which death occurred was as early as thirty-seven. It is worth while to have that fact before us, if only with reference to prognostic uses, as in the case of life-office work, though it may have also a bearing upon general prognosis. My own experience has led me to think that, if a person is once tainted with visceral syphilis, his future is a short one. I do not wish to produce any melancholy effect upon the mind of the meeting, I was only thinking of my patients, who are always in my mind. When once visceral syphilis has declared itself, as Dr. Buzzard has mentioned, sooner or later, or rather sooner than later, the end must come. This experience, however, is an experience obtained by the treatment of the disorder only by iodide of potassium, or nearly only. Latterly I have found, in dealing with visceral syphilis, that one can temporarily relieve the disease by iodide of potassium; but that a mild course of mercury is a good

thing to effect a cure. If that be the case, it seems to me to have a certain bearing upon the question whether syphilis is one and indivisible, or whether it is to be divided into two distinct things. When I was first taught about syphilis, one of the distinctions between so-called tertiary and so-called secondary syphilis had reference to their medication. We were taught that iodide of potassium was the proper remedy for the tertiary stage and that mercury was the proper remedy for the secondary stage. It was almost the only main point in regard to the practical distinction between the two. Indeed, as to all other points of distinction I was rather of Mr. Hutchinson's opinion and thought they were comparatively trifling. Mr. Hutchinson is supposed to take quite a different view of the subject from that which he really holds. Let me read what he says, speaking of the differences between secondary and tertiary syphilis: he says, "Whilst, however, their similarities are marked, so also to some extent are their differences." Then he proceeds to state those differences; and with regard to the second one he says, "The second difference is that a spontaneous tendency to resolution of the new growths and to absorption is constantly witnessed in secondary syphilis, whereas it is exceptional in all tertiary products. It is, however, by no means certain that spontaneous disappearance does not often occur in the case of tertiary growths. Still, it may be fairly granted that a proneness to persist to grow, to spread, and to contaminate adjacent parts, is far more frequently witnessed in the tertiary than in the early formations"—thus putting the thing in a sort of contemptuous way upon its legs again. As to the other criterion, he says, "First, and by very far the most important of the differences between the stages, we place one which has been already mentioned, a tendency to general and symmetrical development in the secondary stage, and to local, restricted, and unsymmetrical formations in that of sequelæ." But listen to what follows:—"This, however, after all, is merely a question of abundance, and not so much of character." It seems to me that when Mr. Hutchinson has said that the difference between the symmetrical and unsymmetrical eruptions, after all, is only a question of abundance, and not of character, he surrenders the whole position, and that Dr. Wilks was right in claiming to belong to the same school of thought in that matter as Mr. Hutchinson. I do not think all has been said about symmetry that may be said. It is admitted on all hands, I think, that secondary syphilis comes out in

a burst of hundreds, if not thousands, of blotches; and since it is the usual experience that tertiary manifestations are one, two, three, or several, it seems to be a matter of common sense that a symmetrical figure like ours would, if blotched on a thousand places, be somewhat alike on the two sides. I will mention an experience that occurred to me last Sunday. I was walking out with a friend, and I had an umbrella, but he had not. Before we got very far I cheered him up—for he was looking glumpy—by pointing out that his figure was symmetrically spotted with rain. He was not a pathologist, and he did not seem to see the joke. I then pulled down the umbrella and showed him the radial symmetry which it exhibited—the beautiful radial symmetry of the rain all round in the radial segments of the umbrella. He had not been bred in the mysteries of fluid pathology, and was rather out of temper and was rude enough to say "Stuff!" He thought the symmetry was the work of the umbrella maker, and not of the rain. Well, there is something in that. One would expect that you would be pretty much alike on the two sides in a rain-shower or in a scatter of syphilis over the surface. You would be symmetrically spotted, not, as Mr. Hutchinson rather satirically says, with a Dutch gardener's symmetry, although I was a little taken aback at finding a mind of Mr. Hutchinson's precision thus slighting exactness—not the symmetry with which nature gardens the two sides of one's face so Dutchly equally—but a symmetry sufficient, shall I say, for the purposes of poetic pathology. I may call this the fallacy of universality. But I shall be told that this does not apply to the really singular and beautiful manifestations of symmetry which Mr. Hutchinson mentioned when he pointed out the symmetry of secondary tonsillitis, of secondary iritis, and of secondary keratitis—that is, if keratitis is secondary, though as it may come in the thirty-fifth year of a man's life it is very late; and it struck me that if Mr. Hutchinson allows a secondary at thirty-five he is hardly in a position to wonder that the tertiary does not appear in the bodies of persons with hereditary syphilis, seeing that if secondary comes at thirty-five, then when the tertiary comes I should think the patient would be dead and safe. Assuming that iritis, keratitis, and tonsillitis are really secondary and also symmetrical, I admit at once that what I call the fallacy of universality would not apply. But something equally simple does. It strikes me that the reason why keratitis is so symmetrical is because there is no more cornea to be affected. If instead of two

eyes we had five, like the scorpion (if the scorpion has five), or if the Society can imagine a syphilitic butterfly with twenty-five thousand eyes, it would have twenty-five thousand patches of keratitis. Or can the Society follow me in this flight?—Suppose that poet whose works I once read who was so struck with the beauty of nature that he wished he was all one great eye that he might see at will through every pore—suppose that poet misled into a *faux pas* (for poetic feeling produces no little of the reality as well the pathology of syphilis)—suppose that the unfortunate monster caught syphilis—then I suppose his universal cornea would be liable to a universal keratitis. In short, it seems to me that the case of the cornea is much the same as the case of the liver. It is true the liver is all in one piece and the cornea in two; but if you are not surprised that the liver is universally affected, what occasion have you to be surprised that the cornea, which happens to be in two pieces, is also universally affected? I name that fallacy the fallacy of totality. Then there is another fallacy in this discussion as to symmetry, but the display in which it appears is so bright that I approach the distinguished authority opposed to me with a great deal of diffidence and with an explanation as simple as "a smooth stone out of the brook." Sir James Paget brought forward, as instances of asymmetry, the suppuration of a rib and the inflammation of a vein after typhoid fever. Sir W. Jenner has pointed out that the typhoid fever eruption itself is not symmetrical; so unsymmetry of so-called sequelæ would not be surprising. Sir James also mentioned the neuralgia of ague. But it is surely on the face of the question that, if you have only suppuration of one rib, it cannot be symmetrical; it takes two ribs to make it symmetrical. Again, if there be inflammation of a vein of only one thigh, you cannot have it symmetrical; and as Sir James Paget did not show any reason why there should have been two attacks and not one, and as the question of there being more than one precedes the question of how they should be arranged if they were present, I think we may say that the question as to symmetry is not even approached in such instances. I call that the fallacy of unity. A gun may be wonderfully well loaded and you may be a good shot, but if you fire out of range you are sure not to hit the mark. My next proposition may perhaps surprise some members. The most remarkable examples of symmetry are in tertiary syphilis. Here, again, I shall be able to bring some facts for Mr. Hutchinson. The first fact will be a case at present under Mr.

Clement Lucas, my able colleague at Guy's, in the person of a woman who has a strictly symmetrical pair of tertiary ulcers, one outside each ankle. He has also a case in which there is a strictly symmetrical pair of ulcers, one on the top of each knee; and also a case where, four years after the infection, the palms of the hands are the subject of symmetrical psoriasis. Then there was a very remarkable case inspected by Dr. Goodhart, of a woman who died of brain syphilis. We found on the pneumogastric roots on each side, where they are in union with the lateral horns of the fourth ventricle, a mass of tertiary syphilitic gummata about the size of a broad bean; it was a most curiously symmetrical development in tertiary syphilis. In those four cases no doubt the fallacies of universality, totality, and unity, do not apply; and I should challenge any one who believes in the exceptional symmetry of secondary syphilis to bring forward facts similar to those in regard of it. I can get more, if he does, to which those fallacies do not apply. These are examples of a true symmetry. They belong to a set of examples of symmetry due to local peculiarities of exposure, &c., some of which were brought forward by Sir James Paget, though they seemed very curious as examples of blood-disease. Pityriasis versicolor was slightly mentioned by him, for instance; but, of course, this is no blood-disease any more than itch is a blood-disease, which is also truly symmetrical; so, too, is trichiniasis curiously symmetrical, and it is not a blood-disease, although the trichinæ no doubt get a lift in the blood as you may in a tramcar. I now pass on to the second point which has been in everybody's mouth who has spoken on the subject—Mr. Hutchinson's great theory as to the relation of the secondary and tertiary stages. This discussion is one worthy of the Society, and I trust I shall not be thought trifling with your time if I take a moment or two longer than my proper share. It will be seen that the division is a great one. We have had one of the most eminent and accomplished surgeons in this or any other day state views in this Society which are directly opposed to no less an authority than the Royal College of Physicians. I see Dr. Pitman before me, and he will no doubt correct me if I am wrong when I state that the College of Physicians is dead against Sir James, because in their nomenclature syphilis is put, not in Class A, general diseases of the blood, but in Class B, of which I do not pretend to give a definition, perhaps Dr. Pitman will. I may now be permitted to state my own view of the

question whether syphilis is a blood-disease. I do not believe or accept the belief that syphilis is ever a thing of the blood especially. I do not mean to say that blood from one syphilitic person will not affect another. In that case it must be *in* the blood. But then we may catch a physician in a railway train, yet you would hardly say that medicine belongs to railway companies; it is merely a matter of transit. I now pass on to the theory as to the relation of the secondary and tertiary stages:—"The importance of all facts or arguments for or against the belief that the gummata of the tertiary stage are purely local and result in renewed growth in formation left over from the exanthem stage." Mr. Hutchinson does not seem to have the remotest conception of the possibility of any other belief. I have a distinct other belief, which I hope to be able to show the Society is capable of being held in a reasonable way. First of all I ask the Society to allow me to state my belief that syphilis is never a thing of the blood only; and my next point is that all febrile disorders, when they strike virgin ground, rage with comparatively great freedom. I will only mention the development of smallpox and measles among races new to it, and bring forward the fact that the powerful and muscular suffer most from the fevers, and that most febrile disorders give immunity, which we may interpret simply by saying that when once people have gone through the febrile course they are no longer susceptible to its influence. I agree entirely with Mr. Hutchinson in the view that syphilis is a fever. I am not sure that I am right in the impression that rests on my mind, but I thought it was Linnæus who first made out that point. It would be well if it were, because botany affords a beautiful parallel for it. I read in the 'Linnean Transactions' nine years ago of a certain palm-tree of the Seychelle Islands (*Lodoicea Seychellarum*) which takes four years for a bud to form, two years for it to blossom, and three years to perfect the fruit. The blossoming is slowed, and in the same way we may say that syphilis is fever diluted with time so as to be cooled and slowed. If you could foreshorten it, as in the case of those photographic puzzles, you would get it out as a fever. I say syphilis is admittedly a fever. It will follow the same laws as the rest of the fevers; and the great law of fevers in reference to liability to attack is, as I have said, that what is fresh is liable to attack. The next point is this. When a woman, years ago, has had syphilis, her syphilised tissues have been so changed that they are no longer vivid to syphilis, and when she gets a fœtus

(a new mass in her not yet syphilised) the independent growth of new stuff is vivid to syphilis. Thus we find that she carries within her, years after her own syphilis, something which has not had anything to do with syphilis, and is ready to take it on. I do not go with so much faith into the history of those ladies Dr. Broadbent spoke of as he has done. There may have been something in the past which they have forgotten. This new lump, the fœtus—which I have heard called osteo- fibro- sarcoma, to all intents and purposes it is a new piece of flesh not yet syphilised, and it takes on syphilis. The placenta is always found diseased in syphilitic abortions. Those who have followed Virchow and gone into this question will say it is not tertiary syphilitic gumma that you get in the placenta. Virchow makes a strong point of that, and says that he has rarely seen tertiary syphilitic gummata. So much the better for my theory. It is said that what you get is fatty degeneration? Pray, sir, what is that? The placenta has no cornea, has no tonsils, no iris; and it cannot have keratitis, or tonsillitis, or iritis. What can the poor placenta have but fatty degeneration? Secondary syphilis of the placenta shows itself in fatty degeneration, and I say this new lump is syphilised and turned out. That will show pretty well why persons apparently free from syphilis should get a syphilitic fœtus. I may be asked how tertiary syphilis comes in adults; and here I shall ask the members of the Society to go back to long-buried knowledge, which, perhaps, they never perfectly appreciated, to knowledge which I remember taking in almost with a sensation of anger. I had read the account in Quain and Sharpey of the histology of the skeleton, and got through the lacunæ and canaliculi and Haversian systems and so on, and then I learnt that Messrs. Tomes and De Morgan had discovered Haversian spaces, and I was very much disgusted. These Haversian spaces were hollows cut in the adult bone—it seemed most unnecessarily—and then filled up with new, solid, good bone. Then I must do justice to my friend and colleague Dr. Fagge, who about three years ago asked me whether I ever recollected that circumstance, and I must say that up to that time I had not revolved it in my mind. He asked me if I thought it possible that the same thing might happen in other textures, for, in fact, the bone shows well written out in its permanent hard inscription what happens in the history of the softer texture; that soft texture might similarly go through all those changes without our being able to detect them. I am happy to accept that view,

because it suits my present purpose. Let me mention a case that I once saw, a case where the right lobe of the liver was gone and the left lobe weighed as much as the whole liver would have weighed ; there must have been an immense production of new substance. A man in training increases the weight of his muscles and bone, he must have new tissue. You see the line of thought that my theory leads me along. Instead of thinking with Mr. Hutchinson that the developments of tertiary syphilis are due to the breaking out again of fires in already effete hearths, I am inclined to think that it comes in such portions of an adult frame as have arisen afresh recently, after the law of Haversian spaces or after the law of hypertrophy, into existence. They have not had their share of syphilis, and are bound to take it in their turn, just as the syphilitic fœtus is so bound. Whether it be a true view or not I do not mean to pursue it before the Society; but I will say that the more I have pursued it the more it seems to fit in with many phenomena which raise difficulties in the minds of those who take the fluidist view. I am not fond of hypotheses, and I am at least willing that if Mr. Hutchinson will agree never to say anything more about his theory I will agree never to say anything more about mine. I shall be happy to hand over to him any few facts which I have been able to collect before the Society, feeling sure that he will be able to fit them into the construction which he will finally crown at the conclusion of our individual efforts.

Adjourned Discussion, March 21st.

Mr. Thomas Smith.—Mr. Hutchinson very early in his remarks stated that to the annoyance and misery of thousands syphilis did not always end with its apparent death at the end of the secondary stage. But, later on, he remarks : "the lesions of the tertiary stage by their non-symmetry appear to prove that now at least the blood is not concerned ;" and as additional evidence in favour of this absence of blood taint in the tertiary lesions he cites their non-contagiousness and the power that is exercised by local remedies in their treatment.

Had Mr. Hutchinson himself attached any real importance to this want of symmetry in the lesions of the tertiary stage in contradistinction to a supposed symmetry in the secondary symptoms I

should have been prepared to dispute altogether this alleged contract between the two sets of symptoms. But since he subsequently admits that "symmetry and no symmetry is a question of abundance and not of character," it is not necessary to discuss this question: since we should all agree that in the early stage of constitutional syphilis the symptoms are more general and more widely spread than in the later stage.

Concerning the non-contagiousness of the lesions in the tertiary stages Mr. Hutchinson admits that there is no direct proof one way or the other, and as I do not intend to take my stand on this point I am willing for the sake of argument to allow that secondary syphilis is non-contagious. For the same reason I will acknowledge that local remedies have some efficacy in the treatment of the tertiary syphilitic lesions he enumerates: viz. palmar psoriasis, lupoid tubercles, some affections of the tongue. Yet I cannot allow that it is usual to see any permanently beneficial result from local treatment in ulcerated gummata, syphilitic ulceration of the legs, in deep tertiary ulcers of the face and nasal cavities; yet in all of these the remedy can be brought into contact with the diseased part otherwise than through the medium of the circulating blood, yet it is only in this latter manner that a cure can be effected.

But what can be said in favour of the view that the symptoms of tertiary syphilis are as a rule the result of a persistent blood contamination? Passing by all that has been urged on this point by others, I would take my stand on a fact that is not disputed by Mr. Hutchinson, namely, that the power of hereditary transmission persists during the tertiary stage; or as he puts it, "the risk of hereditary transmission persists long after the cessation of blood contamination."

Mr. Hutchinson admits a "difficulty in conceiving of this;" to me the difficulty is insuperable in the case of syphilis.

Now, I assume that the terms "blood contamination," blood disease, are employed in their generally accepted sense, and that Mr. Hutchinson means that a man may beget syphilitic children when he himself is no longer the subject of constitutional syphilis. This, I take it, is the plain meaning of Mr. Hutchinson's statement, and this is what I cannot for a moment admit.

I may at once be met by the objection that there are other constitutional diseases, such as gout, which a man may transmit to his offspring, he himself being free from the disease at the time of trans-

mission. Or still more—a man may transmit to his son a something which may give no sign of existence until it becomes gout in the grandson; so that in this last case the intermediate individual (the pestiferous person) transmits a poison or disease that he never at any period of his existence suffered from.

I have taken gout as the strongest instance that can be brought forward in support of the view that a man may transmit a disease to his children, and yet not be himself the subject of that disease.

Now, in relation to the heredity of gout, I believe it can be said with more truth than it can of syphilis that "the germ or sperm does give as it were a potentiality of development in one definite direction which potentiality shows itself at variable periods of life"—these are Sir W. Jenner's words.

This potentiality of development transmitted with the sperm in the case of gout requires years to breed the fully developed disease, for a man generally grows up into gout or, perhaps, more often down into it, and indeed it may require more years than there are in one man's lifetime, for in the case where gout survives a generation this potentiality takes one whole lifetime and part of another to produce the actual disease.

In strong contrast to all this are the circumstances attending the hereditary transmission of syphilis, I maintain that from the earliest contact of the parental sperm with the ovum there is distinct evidence of the existence and transference of a deadly disease and what I take leave to call a blood disease—a disease which may destroy the fertility of the seminal fluid, may blight the ovum, may kill the fœtus in utero—may attack the newly born infant, may poison the mother through the fœtus, in her pregnancy or during parturition.

In support of these assertions it is not necessary for me to do more than mention the notorious infecundity of syphilitic marriages, the abortions, miscarriages, and other catastrophes which may occur.

In all these particulars there is a striking contrast to what occurs in gout, nor will the same explanation suffice. In syphilis the germ or the sperm transmits something more than a potentiality of development; it transmits a real disease directly continuous from parent to offspring.

I would direct attention to one or two other points of difference between the two diseases: hereditary gout may miss one generation and appear in the next; does hereditary syphilis ever do this? Who ever heard of a mother contracting gout by bearing in her womb

the child of a gouty father? Who has not known of syphilis communicated in this way; and how is the disease passed to the mother? through the blood of the fœtus: in the placental villi to the blood of the mother in the uterine sinuses. If this be not a blood disease in the reasonable sense of the term, what is a blood disease? It is an all-pervading disease.

I am very anxious that Mr. Hutchinson should not misunderstand me. I quite agree with him that the degree of blood contamination is constantly diminishing, in syphilis, until it comes to a vanishing point, that the risk of hereditary transmission in the later stages of tertiary syphilis is very small; that it is possible for palmar psoriasis, ulcers, and other lesions considered evidences of the disease to outlast the stage of blood contamination. Yet I want to persuade him that the risk of hereditary transmission does not persist long after the cessation of blood contamination; but that the power of hereditary transmission is evidence of the existence of blood contamination.

Mr. Hutchinson concludes his paper with this inquiry—Are there any facts which favour the belief that syphilis continues to be a blood disease after the cessation of all tendency to produce symmetrical symptoms? My answer to this would be, Yes,—there are many recorded cases where parents suffering from gummata, caries, and the later lesions of tertiary syphilis, have begotten or given birth to children who speedily became the subjects of the same disease in its secondary form.

Sir William Gull.—I rise to speak on this important subject, because of late years the branch of the profession to which I belong has become greatly interested in the question of syphilis. For too long a time, I think, syphilis was entirely consigned to the tender mercies of the surgeons; but I confess, among the things I have seen and studied in medicine, the history and course of syphilitic infection has seemed to me one of the most interesting. When we consider the origin of the poison (which is no doubt associated with the generative fluids), we might almost predicate of it that it would be a strangely pervading poison. That may not, perhaps, at first strike those who hear me; but I think, if you consider the origin of the poison, you will say that it is most likely we should have predicated of it that it would pervade almost every tissue of the body, and that it would not be limited to the individual, but be

propagated to his offspring. In that respect it is always peculiar. It has always been an interesting point with pathologists to make syphilis one of the fevers. Dr. Robert Williams, in his book on poisons, classes it with typhus and typhoid; and those who have thought most on the subject have regarded it as belonging to the class of fevers. But, though it may be so placed, it is still a peculiar poison. Take the case of variola. A father has variola, a mother has variola, but the children of those parents seem to be quite uninfluenced by the variolous process. That is not the case with syphilis. The same may be said in regard to typhoid, typhus, measles, scarlatina, and, in fact, all the class of the so-called fevers. Therefore, if we are to place syphilis amongst the fevers, it is certainly a peculiar fever in this respect, that it is not limited in time as other fevers are. Mr. Hutchinson said in his paper that he thought that, if syphilis were left unhindered by treatment, it would be limited in time. I agree with him that in some respects it is limited in time (I have myself noticed such cases) when uninfluenced by treatment, but that must be in a particularly healthy individual, probably young in life and unspoiled by those other divinities which go with Venus, namely, Bacchus and Mercury. Occasionally it is limited, and most remarkably limited, in time; Nature herself can stamp it out; but that is by no means its universal history. If it be placed among fevers, it is a fever not limited in time as they are, for it can go on, I think, almost *ad infinitum*. We have evidence that a father may beget a syphilitic child when he is past the constitutional condition of syphilis, as has just been insisted on; that a child may be born apparently healthy and may remain healthy for some weeks, and yet after a time, if children are vaccinated from it, they may have syphilis. We have nothing like that in ordinary fevers. A child born of variolous parents is as free from the influence of variola as though they had never had it, and there is no mode, so far as I know, of producing any influence upon it. Again, it is a fever without pyrexia. The febris and the pyrexia have, I am happy to say, long been dissociated in clinical medicine. Then, is it a blood-poison? We have come to the old question of blood-poison again, but I will not take it up. My friend Mr. Smith has just said that he uses the term "blood-poison" in a general sense just as people commonly say "it is in his blood." Then he adds that he thinks it is in the blood proper, that is, in the circulating fluid, and not in the tissues and not in the lymph. He first uses

the term in a general sense, and then in a more or less limited sense. Well, I think syphilis is a flesh-and-blood fever; I think that is its whole clinical history, and that that is the proper expression of syphilis. There is no tissue in which it does not exist; there is no fluid in which it does not exist. We have a proof of its being a flesh-and-blood fever in this, that an apparently healthy child may be born of syphilitic parents, may be vaccinated, and the vaccine lymph may pass from one child to another without conveying syphilis; but, if blood is mixed with it, it has an influence. Mr. Simon will tell us whether that is a fair inference or not. If so, then syphilis continues to be a blood-poison even in healthy children, so that it is a flesh-and-blood poison; it is a flesh-and-blood fever. Then, as regards the tertiary effects, which physicians have most to do with. Surgeons have the primary disease, and largely the secondary disease, and then physicians come to those singular tertiary effects; and there I think I must agree with Mr. Smith that it still continues to be a constitutional affection. I think that syphilis continues to be a constitutional affection through the whole life of the man who has had it. Syphilis once, syphilis ever; syphilis general, syphilis universal, in the man all the time he lives. He may wear it down more or less, still it may remain there. This is very important clinically. You may have a syphilitic patient who may give no evidence whatever of syphilis to the most experienced person. Now, there is no better authority on this matter than my friend Dr. Wilks, to whom we owe a great deal of our knowledge of the effects of syphilis upon the internal organs. I remember a case which I have often quoted, because it shows how little evidence we can have except local conditions to guide us in regard to the after-effects of syphilis. A patient was placed under my care whose nutrition had become extremely faulty; he had lost two stones and a half in weight, and had become more or less generally paralytic—quite different from the ordinary limited form of paralysis to which we are accustomed, as depending upon deposits of gummata. I knew that he had had syphilis, and that from time to time he had had affections connected with that taint. He became very ill and went into the country, where I went to see him, and prescribed accordingly. I was unable to continue my visits, and Dr. Wilks went to see him for me; and he, though he is so apt at all this pathology, could not see, and would not believe, that the man was suffering in any way from the effects of syphilis. If the result of treatment be a proof,

he certainly was suffering from it; for directly he was put upon proper syphilitic remedies, he not only recovered his weight, but his health, and lost all his nervous affections. I believe, therefore, that the effects of syphilis continue, or may continue—I do not say that they actually do—during the whole period of life. Hence Mr. Hutchinson's difficulty, I think, is one that he rather creates for himself. In that respect I agree with Mr. Smith. We see the effects in the hair and in the complexion; we smell it in the odour of the sweat, and that when there is no local development of syphilis. I remember a case of this kind. A man was supposed to be fading away from tubercle; it might be diffused, but where nobody seemed to know; and he was sent to me for examination. I found no tubercle, but I was struck with the odour of the sweat; and I said to Sir James Paget, "I think I can smell tertiary syphilis." We found no gummata, no psoriasis, nothing to show the presence of syphilitic poison in any tissue; but I believe it was there. I remember remarking to Sir James Paget: "If we had found a copper-coloured rash in his palm, we should have been satisfied." We found it nowhere about him, yet the man was syphilitic. This was years after he had the taint. He was immediately put upon the proper remedies for the tertiary taint, and he quickly recovered. I believe, then, that over and above the gummata there is a general condition still remaining, quite bearing out what Mr. Smith has said in reference to there being no limit to the affection of syphilis in the tissues and in the blood. I cannot sit down, occupying the position I do in the profession, without thanking Mr. Hutchinson and those who have worked with him in this matter. I think the whole profession of England are greatly indebted to Mr. Hutchinson, to Dr. Wilks, Dr. Buzzard, Dr. Broadbent, and a number of other gentlemen belonging to this Society who have worked out this matter. I do think it is an enormous advance that we have made in recognising that there is one poison which produces an infinite variety of effects according to the constitution upon which it operates. We have also made a great advance in believing that what are called primary, secondary, and tertiary stages, are artificial divisions, rather than divisions that nature makes. We have learned, I think, in these days, that it does not require a primary sore to infect a second person; that the secondary disease is as infectious as the primary, and is only a general form of the primary; and that in all likelihood the tertiary disease is but a

diminished condition of the secondary, as we see by the children who are born of such parents.

Mr. Simon.—I rise, sir, in answer to your summons; but, indeed, for many reasons, I would have much preferred to remain merely a listener to this very interesting discussion. On the subject about which Sir William Gull referred to me I have nothing to say; but there are some, in fact there are numberless, parts of the argument which have interested me very much, and if I had expected that you would have done me the honour to call upon me, I would have made a note or two of the points on which I might offer some remarks. But generally, perhaps, I might offer this criticism on Mr. Hutchinson's admirable paper (in the admiration of which, expressed by Sir William Gull, I most entirely concur), that I think it calls upon the Society too much to answer with an absolute "Yes or No" questions that are really to be answered by "more or less." As to the first proposition, that syphilis, when it ceases to be a blood-disease, ceases to produce symmetrical effects, I should demur somewhat to the strictness, the hard-and-fast line, of that statement. In the first place, I accept entirely Sir William Gull's phrase as to syphilis being a flesh-and-blood disease. Amongst the class of diseases to which syphilis belongs, I do not know any pure blood-disease. Syphilis is more or less a blood-disease at different moments of its existence. At one part of its existence it is, of course, not only a flesh-disease, but also in a very intense and abundant degree a blood-disease; the contagium is plentiful in the blood; and that is a period in which all that comes from the body is infective: in which the sperm is infective, in which the secretion of any ulcer would be infective, in which the catarrh of the uterus and vagina would be infective, in which vaccine lymph taken from a child would be infective, and so forth. But, I apprehend, there is no abrupt line to be drawn between that condition and the condition of a patient with tertiary syphilis. The quantity of contagium in the blood gradually diminishes, and the power of it also gradually diminishes. As Sir William Gull capitally expressed it, there is here the strong contrast of the acute fevers. This is a disease of indefinite duration. As the quantity of contagium in the blood diminishes, a time comes, no doubt, in which it is a chance (but this is a question only of quantity) whether a particular secretion will contain contagium or not. In regard to the early stages of syphilis,

it is a tolerable certainty, and often I suppose an almost absolute certainty, that the patient will have syphilitic offspring. As he goes on, it is no longer a certainty; it is a chance; but that, I apprehend, is a question of quantity. He may have some syphilitic and some non-syphilitic children, but the successive children which are born syphilitic will as time passes be less intensely syphilitic. The probability of his infecting his wife diminishes even more rapidly. But in neither respect is there any abrupt line of difference; there are not two distinct stages of diseases. As regards the gummata, there again is a point where I would raise question as to the hard-and-fast line that Mr. Hutchinson, in some of his propositions, seemed to draw. He seems to speak of gummata not as directly syphilitic phenomena, but as new phenomena arising in parts which the syphilis, in its secondary stage, has affected: as sequelæ of syphilis, but with syphilis no longer effective in them. Well, I do not understand the pathological possibility of (in that sense) an active sequela; I understand a passive sequela, I understand a cicatrix; but when anybody talks of a morbid *process* as the sequela of a given cause, that means that the cause is still operative. In so definite a process (a process quite peculiar) as the growth of gummata, I cannot conceive the "sequela" otherwise than as representing the continued operation of the original cause. I do not differ from the positions which Mr. Hutchinson has taken in his paper, if the phrases are a little modified; if, instead of calling upon us for yes or no, he allows us to answer "more or less." I agree that, in proportion as secondary syphilis ceases to be an infective disease, the symptoms which it produces are likely to be unevenly distributed on the two sides of the body, as was explained by Dr. Moxon at the last meeting of the Society; but I do not regard that as a very important fact. I agree that, in proportion as one arrives at the period in which gummata develope themselves, in proportion as one gets the tumour-producing power of syphilis exhibited, one has evidence of a condition of the poison, probably a weakening of it, in which it is not likely to affect offspring. But, I confess, I do not think it would be safe in practice to take these as absolute propositions, and to say that, because a man has gummata, that man will not transmit syphilis to his offspring, or that, because a man's syphilis is unsymmetrical, he will not give syphilis to his wife. It would not be safe to take either of those propositions. I think Mr. Hutchinson's propositions may

be taken as approximations, but not as mathematically exact propositions. As regards a matter that has been adverted to this evening by Mr. Smith, the hereditariness of syphilis as compared with the hereditariness of gout, I think the two sorts of hereditariness are quite different. The hereditariness of gout is like the hereditariness of a Roman nose; it is part of a family likeness; the particular chemical type, the particular mode and rate of the chemical change of tissues, passes from the father to the son as the shape of features passes. But, with regard to the transmission of syphilis, the hereditariness is of a different kind; it is more like that which one has in comparative pathology, for instance, in some of the silkworm diseases; it is like the passage of a parasite in the ovum from one generation to another. It is not the case of the law of development inherited from one to the other, but the case of a material something passing from one to the other. Regarding the syphilitic contagium, in the present state of knowledge, it is no great assumption to suppose that it is an organic something, a growing something, a multiplying something; you have that something passing from the parent to the ovum and vitiating the development of the ovum. A question of much interest raised in Mr. Hutchinson's paper is as to the power of syphilis to modify other diseases, and particularly as to its convertibility into scrofula. A consideration which I think an edifying one in relation to this question is, that the diseases into which syphilis is most accused of transforming itself are not the diseases which are most definite. The Pathological Society, with its accurate spirit of investigation, has not yet, I believe, succeeded in defining scrofula. Tubercle we have got to know something definite about; but scrofula, as a disease to be distinguished from tubercle, is but an unknown quantity for comparison. Be that, however, as it may, I have no knowledge or experience of syphilis either producing any other disease than itself, or being capable of modifying other diseases, though, of course, it may mix its phenomena with theirs. Here, too, I would qualify a remark made just now by Sir William Gull as to the variety of effects that syphilis produces in different subjects. Variety of degree —yes; but variety of effect—I venture to question. Syphilis is as definite a disease, I believe, as any disease known to our nosology. Although you have in different individuals great differences in intensity of result, I do not think you have any other great difference. That sort of difference of result you get in relation to all infective

diseases. Scarlatina, smallpox, measles—any one of these diseases may, as regards degree, show itself as differently as can be in one family from what it shows itself in another. And so, if you take a hundred cases of syphilis, no doubt you get very great differences of result; but those, I submit, are mainly differences of degree, and not differences that admit of being described as nosological varieties. As to the question raised by Mr. Hutchinson, whether gummata are deferred manifestations of a change of tissue affected in the second stage of the disease, I have expressed some hesitation which I would beg leave to repeat. It seems to me as more probable, looking at the whole history of syphilis, that gummata result from the weak action of a contagium which has been present in the body from the first, than that the tissue has all along been changed. The contagium may possibly have been there from the first, but, at any rate, has become weakened. As we see other evidence of the contagium weakening in the body affected, as we see it in relation to hereditary transmission, so here, where the contagium has been lingering in the tissue or in the body for a number of years, and presently produces its result, we should expect the result to be of low intensity; and it is only in accordance with what one knows in pathology that the irritant (if one may call it so) of this contagium, the irritant which in its stronger dose would produce an inflammation, would produce necrosis of tissue, in this late stage produces only hypertrophic phenomena, eventuates in tumour-formation. But if this question is to be well examined, it must be examined not from theory, but by fact. The aim should be to examine as many cases as one can get of secondary syphilis; and though it is very seldom that one can get for examination the dead bodies of adults with secondary syphilis, the opportunities of examining children who die with congenital syphilis, which is secondary syphilis, are innumerable, and they might be advantageously utilised for the Society. Examination of their tissues and reports on them to the Society would be of great value. Then, as regards the ulterior effects of syphilis, very important knowledge may be contributed in the matter of family history; and here almost every member of the Society, particularly those who are engaged in family practice, have opportunities of giving great assistance. Any one who has for long periods of time whole families under his observation is able to trace the true outcome of syphilis, and to see as a matter of fact whether other given diseases are less or more frequently produced—tubercular

diseases for instance, and bone-diseases, in the offspring of syphilitic families than in others. As to whether syphilis itself ever appears in more than the next generation, I may say that I am myself aware of one case where very strong suspicion was felt that syphilis had so manifested itself; that is, where a lady, whose father had died of tertiary syphilis, herself had a first child that was judged to be syphilitic, and had in her own person some symptoms which were ascribed to the same poison, there being not the smallest possibility of syphilis, as far as could be ascertained, coming to her in any other way than by that of descent. One would of course not venture to form an opinion on a single case, but these are matters in which very exact observation requires to be made, and in which very valuable material could be contributed by those who would keep accurate records of family history.

Sir William Gull.—Let me offer a word in explanation with regard to what Mr. Simon has said as to the difference of effects or varieties of effects. What I wished to convey to the Society was this:—I said I thought that we had made a great gain since we had determined that there was one syphilitic poison producing a variety of effects according to the constitution. One person, for instance, has roseola, another has a scaly rash, another a papular rash, another a pustular rash. These are varieties of effects that were supposed to depend upon varieties of poison, and I wished to assert that we had made a great advance in believing that the variety of effects was due to the variety of people affected, and not to the original poison.

Mr. Thomas Smith.—Permit me to say that Mr. Simon is mistaken in supposing that I compared gout to syphilis; I endeavoured to contrast them.

Mr. Simon.—Quite so.

Mr. John Wood.—Since these debates were first begun I have learnt a great deal. First, I have learnt the presence among us of some very old opinions which I am astonished to see crop up again. The old debates between humoralists and the solidists, one would have thought, belonged to the legendary history of the profession, but the discussion seems still to prevail. I am glad that most of the speakers, in fact, are agreed in the opinion that syphilis and other diseases affect the constitution, affect the whole body, the blood, the lymph,

and the solids. The definition which Sir William Gull has given us, like those which he so frequently gives, is a happy compendium; the term flesh-and-blood disease is certainly far better than the term blood-disease, though it scarcely covers the whole ground. It seems that this idea has also resulted in another phenomenon, which I am surprised to find is yet existent. There are persons still amongst us who believe in the multiplicity of poisons; at any rate, in there being two or three kinds of venereal poisons. I had brought myself to believe that the poisons of syphilis, the manifestations, whether primary, secondary, or tertiary, were simply the resultant between the virulency and the amount of the dose of poison originally imbibed, and the opposition of the system to the admission of the poison. In the case of a primary sore it is the admission of the poison into the blood primarily producing, in one case, indurated chancre, and, in another, soft chancre, and so on. In the case of secondary infection, where the disease passed from the blood into the solid tissues, the skin, for example, or the mucous membrane, we have another compromise or resultant between the resistance of the healthy tissues and the aggressive influence of the poison. We see the same in the infliction of wounds and various injuries which are more purely surgical. We see, in some instances, an amount of solid fibrinous material thrown up as if a foreign body in the tissues; in other instances it softens down into suppuration. You have here the difference between a hard and a soft chancre, or you have potentially the difference between a dry rash and an eruption results in the formation of fluid matters. I will now pass on to the point of the tertiary symptoms being sequelæ, or a continuation of the action of the syphilitic poison. I am decidedly of the same opinion as that expressed by Sir William Gull and Mr. Simon, that there can be no definite line of distinction drawn between these stages, and I think Mr. Hutchinson has done wrong in endeavouring to define such a line. He has fallen into the same error which many other syphilologists have fallen into in endeavouring to make a distinction between different kinds of poisons. So far as I understood Mr. Hutchinson, he seemed to agree with Dr. Wilks that all tertiary lesions were laid down during the time of the secondary fever, and that afterwards they simply followed the laws of ordinary growth. If this be the case, it seems to me that Mr. Hutchinson laid a little too much stress upon the symmetry of secondary diseases and the asymmetry of tertiary lesions. If the

sequelæ of simple growths be foundations laid down only during the secondary fever, we must also acknowledge that a patient while labouring under tertiary symptoms will not beget a syphilitic child, and will not be able to communicate the disease to another. Although Mr. Hutchinson and other speakers have alluded to the case of a woman in childbirth having scarlet fever, and the child being born covered with scarlet fever eruption—although this may be a transmission through the germ of scarlatina poison, yet, on the other hand, it may be simply a case of infection by contiguity. There is one point which has not been alluded to in the debate. Is it true or not that the races of men are becoming syphilised? Is syphilis milder in its manifestations than it used to be when it was first introduced among the European races? If this be not true, why do we not see the dreadful cases that were first seen on the coast of Africa? It is manifest that they cannot have been entirely due to the abuse or non-abuse of mercury. If syphilis be capable of effecting such a change in the races of mankind so as to render its ravages less virulent, it is quite clear that it will come under the classification of diseases like gout and rheumatism, especially the latter, which in many of its features has a strong resemblance to syphilis. I think this is an interesting point that ought not to be entirely overlooked in the discussion on this subject.

Dr. Robinson.—As regards the nature of syphilis, its unity or duality, I venture to think that the general opinion among army surgeons is in favour of the unity of the disease. The opinion on this point, expressed by Mr. Hutchinson and Sir James Paget, are entirely in accordance with my own. The disease, as it appears to me, depends upon certain conditions of the constitution, on temperament, upon what we may perhaps call the receptive condition of the patient at the time, and further upon the ablutions, if any, observed. I have always considered such circumstances as these as modifying the action of the poison in the system. Many years ago, in a detachment of the Guards in one of the parks, phagedæna occurred in a number of the men. In that case I was impressed with the idea that there are cases in which the virulence of the poison overrides constitutional infirmities. I remember also, in Limerick, a case in which it seemed to act in like manner. There was a perfect epidemic of syphilis there, all the cases being of one type, soft chancre associated with a peculiar indolent bubo. The

hospital was filled with that class of cases. It seems to me, therefore, that there are cases in which the usual mode of procedure of the disease is modified. With regard to hereditary syphilis, I think that the prevalence of hereditary syphilis exists to a much greater extent in private life than in the army. Why this should be the case is, I think, tolerably evident. In private life the condition of the patient is comparatively little known. In the army you have a considerable knowledge of the patient for a long period. Another point is that the sequelæ of syphilitic disease in the army are much less apparent. I should strongly hold to the view that syphilis in regard to its hereditary character is materially modified and reduced to a minimum by an early and judicious use of mercury. The last point to which I will refer is the connection between syphilis and phthisis. I presume that it is notorious to all that in the Household Brigade syphilis is very prevalent, and that phthisis is also prevalent. In a batch of seven or eight men that passed through my hands yesterday, who were discharged with phthisis, four had been under treatment for syphilis. I have felt that the connection between the two diseases is a remarkably close one. Were it not for the existence of several other equally potent causes, such as drink, exposure at night, and the use of a tunic that exercises an injurious effect upon the circulatory system, I should be disposed to think that syphilis was a more immediately exciting cause of phthisis; but it is impossible to dissever one of these causes from the others.

Mr. Venning.—I am anxious to derive as much practical information as I can from this debate, and therefore I would ask Mr. Hutchinson if, in his reply, he will give me a few more details with regard to one paragraph in his paper.

I allude to that where he speaks of the theory of dualism, in which he makes the following statement:—"I think we may say of dualism that it is dead, and that the far simpler creed which attributes the soft chancre to contagion with inflammatory products, produced by syphilis, but not as a rule containing its germs, is the one which now attains general acceptance. We have, then, in syphilis but one malady and one virus."

Now, it is with regard to this "far simpler creed" that I seek for further information. I do not quite understand how a sore can be said to be attributed to contagion with inflammatory products

depending upon syphilis, and yet not containing its germs. Mr. Hutchinson's theory would place that form of sore which has been called by syphilographers the "local contagion sore" under the heading of a common inflammatory sore, for to speak of it as depending upon syphilis, and yet being devoid of its germ, would be like speaking of the semen of the body without the spermatozoa. But surely in this soft chancre we have something more to deal with than a common inflammatory sore. The definite course it runs, the fact that it is always attended with loss of substance, the inflammation of the lymphatic glands running on to suppuration, which so frequently accompanied it, and the fact that the same kind of sore can be produced by pus taken from the interior of the gland so affected, which cannot (in the earlier stage) be accomplished with that taken from the structure surrounding the gland, in addition to its perfect inoculability, and that through a series of inoculations, all tend to show that we have something to deal with of a different nature to a common inflammatory sore, and acknowledged to be different from the true infecting chancer, with all its sequelæ, or that third form of sore which occasionally occurs in persons who have been previously infected with syphilis.

Dr. Farquharson.—I am glad that Mr. Venning has broached this subject; because, although Mr. Hutchinson has told us that dualism is dead, still I venture to think that something may be said for it; or perhaps I may say that dualism never had reason to be considered alive at all. I do not think that any one ever seriously supposed that there were two classes of venereal poison running their course side by side in the human constitution. I see no great reason why we should not consider that the soft sore may be an independent pathological unity, which may, without having any power of affecting the system, be able to carry on its operations in the system in local forms. If we cannot allow that there are two sores of this sort, I think that our difficulties are much increased. Formerly we could tell our patient, with a certain amount of security, that he would or would not suffer from secondary results. Now, however, it is impossible for us to tell him whether in future, after suffering with a soft sore, he will or will not be seized upon by the dreadful ravages of syphilitic disease. With regard to the analogy of the exanthemata, I think we are bound to take advantage of the difficulties which Mr. Hutchinson has himself raised in this matter. I think it

is not by any means necessary that the hard sore should produce secondary symptoms at all. Secondly, we have numerous irregularities in the development of secondaries. We cannot say beforehand what form any one will have. I think Mr. Hutchinson's explanation with regard to the effect of mercury as disarranging the evolution of syphilis, cannot in practice hold good. My experience is that mercury, when administered carefully and systematically, has the effect of rendering syphilis more regular. One point of agreement with Mr. Hutchinson's views regarding the likeness between syphilis and the exanthemata is, that very frequently there are much milder tertiaries after severe secondaries. There is another point, as to the lines of the tertiary disease being laid in the secondary. I can only bring forward one proof of that; that is a case that I brought before the Clinical Society last year, or rather a class of cases in which a certain form of hæmoptysis comes on in relation to secondary syphilis, which, if not checked by mercurial treatment, is apt to run into a tertiary formation.

Dr. Greenfield.—If I venture, sir, to make a few remarks on this important subject, it is because I think that hardly enough stress has been laid on the knowledge to be gained from morbid anatomy in the course of the discussion. It is, I think, to be regretted that, beyond some slight allusion to certain points, there has been but little reference to this side of the question, and that so little light has been thrown on the histology of syphilitic new growths. Certain questions have been raised which can only be solved by an appeal to the data of the *post-mortem* room, such, for example, as the question of the occurrence of gummata in the secondary period of the disease. But there has, I think, been too great a tendency to speak of the gumma as the sole and characteristic anatomical product of syphilis. That it is so in a high degree is beyond question, but it would be to ignore and reject a very large part of our knowledge of the effects of syphilis if we were to accept the term in its common use, and exclude from it those infiltrating forms of syphilitic new growth which Dr. Wilks has done so much to establish as truly syphilitic in their origin.

But it is evident that some part at least of the ground gained in our knowledge of syphilis during the past ten or fifteen years, on which Dr. Wilks has justly congratulated the Society, is in danger of being lost. For example, Dr. Fagge has expressed a

doubt as to the existence of syphilitic infiltration of the lung, in which Dr. Wilks, at any rate, believes. Mr. Hutchinson has spoken of the gumma as the typical product of tertiary syphilis, but he seems to look upon it as solely a form of inflammatory growth characterised by its hardness. I have no doubt that Mr. Hutchinson would employ the term in its widest sense, and would include under it the most minute nodule, and even an infiltration. But to a very large number of persons the term gumma is a synonym of a more or less caseous product; its essence is decay; it is nothing but a mass of small cells, aggregated together and rapidly undergoing fatty degeneration, and either becoming encapsuled in fibrous tissue as an effete product, or capable of rapid absorption. In this sense the gumma may be recognised in a large number of cases by its naked-eye characters in certain stages; but when cretaceous or even caseous, or when we have nothing left but a fibrous scar, we can only judge either by a knowledge of the history or by its locality and distribution that it is of syphilitic origin; so that, as Dr. Wilks long ago showed, we really depend on other features than the external appearance for our recognition of the cause, and it is a question of probability rather than of certainty.

When we come to inquire what are the microscopic characters of gummata, we are again met by the widest diversity of opinion, both as to what is actually seen and as to the relation which the growth bears to inflammation and to other morbid growths, and how far the microscope can aid us in distinguishing them. I will not detain the Society by describing the various views held by distinguished pathologists on this subject. But as regards the distinctiveness of these characters, even Dr. Wilks has said that, though he has from time to time regarded certain features as specific, he has been subsequently led to abandon them.

There are many reasons why it is very desirable that we should ascertain, if possible, what are the characteristics of syphilitic new growth, especially in its earlier stages. An immense number of lesions of various organs are ascribed, and no doubt with good reason, to the syphilitic poison. There is scarcely an organ or tissue in the body which is not, in some form or other, liable to affection in course of the disease. Quite recently the question of the relation of syphilis to the production of aneurysm has been hotly discussed, and is still undecided. Syphilitic disease of the smaller arteries was long ago described by Dr. Wilks, but has recently been

much more fully investigated. Again, it is well known that thrombosis is very apt to occur in syphilised subjects, but from what cause has been long doubtful. These and many other points indicate the desirability of extending our knowledge of syphilitic disease to the minuter elements, and not confining our attention to those changes which are visible with the naked eye. Mr. Hutchinson has raised the question of the occurrence of nervous affections in infantile syphilis; but in order to decide this it is absolutely needful that we should be able to recognise the earliest changes in the nervous centres before the growth of actual gummata. The mere fact of the occurrence of nervous disorder in the course of syphilis is of little value in proving causation.

Even if we consider only gummata, it is evident that what is most important is to trace the course of their development, for at some epoch of their existence they must be minute and recognisable only by the microscope. As often seen, syphilitic gummata are but the extinct craters of volcanoes, perhaps with a few smouldering ashes, but mainly distinguishable by the scoriæ and lava in the form of puckered fibrous cicatrices.

There are, undoubtedly, certain external features by which gummata are distinguished from other morbid products. Now, if it be allowed that these do not depend on the nature of their constituent elements, they must depend either on their arrangement or on their relations to other tissues; that is, just as a building gains its distinctive appearance from the architect's plan rather than from the bricks and mortar. In order, therefore, to ascertain the cause of its special characters, we must study the course of its origin and development, to see whether they present any peculiarities.

I have examined microscopically a large number of organs affected with syphilis, including gummata from various organs, at all stages, and infiltrating growths, as well as diseased arteries, with the idea of throwing some light on this question. And if I venture briefly to state what, so far, have been the points which have seemed to me the most important, I do not in any way pretend to have arrived at the solution of the problem, but simply to invite the observation and investigation of others. It is impossible to describe minutely here the various changes. I shall only briefly sketch them, trusting to a future opportunity to bring forward the specimens on which the statements are grounded.

During the period of their active growth and development, before

they have attained any size or undergone degenerative changes, gummata appear to consist in a measure of a sort of low form of lymphoid tissue, highly vascular, which is composed mainly of rounded or ovoid cells with very distinct nuclei and nucleoli, and a distinct nucleated stroma. If one examines the earliest form of infiltration, *e.g.* in the brain, it is found to consist of a growth of similar nature, extending mainly along the perivascular sheath of minute arterioles, not, however, forming nodular masses, as in the case of tubercle. By the increase and coalescence of these small tumours are formed. Very rapidly, however, there seems to occur thrombosis of the vessels supplying the new growth. This, so far as I can make out, is due to a thickening of the walls of these vessels, partly by growth in the outer coat, but also apparently by growth of the inner coat, which seems in part due to proliferation of their endothelium. This proliferation and consequent thickening of the inner coat can be readily seen in the small arteries of the pia mater surrounding a gumma in the brain, some very characteristic sections of which have been prepared for me by my friend Dr. Saundby. The change in these vessels is identical with that described by Heubner in his recent monograph, as affecting the cerebral arteries, independently of gummata.[1] But it can also be seen in the minuter arteries, not only of the brain, but also of the kidneys. It is, I believe, one of the most important points of distinction of syphilitic from other morbid growths, and accounts for the early necrobiosis of the newly formed tissue. Very speedily, however, either by a further growth of the small round cells, or from the irritation of their presence, a growth of embryonic connective tissue in and around the earlier nodules occurs, this tissue being characterised by the variety and luxuriance of its cells (as Virchow has observed) and by its high degree of vascularity. This growth takes place also along the walls of the occluded vessels, which become converted into fibrous bands containing an immense number of smaller vessels. According to Heubner, a single new vessel may thus be formed in the interior of the old one; but I have only been able to see the formation of a large number of smaller ones. The partial caseation and yellow colour of the gumma appear to be due to the earlier or necrobiotic changes; the firmness, smooth section, and persistence, to the later growth of a highly vascular though dense fibroid tissue.

[1] 'Die Luetische Erkrankung der Hirnarterien,' von Dr. O. Heubner. Leipzig, 1874.

The infiltrating growths do not appear to differ in any marked degree from gummata, save that in them the growth of embryonic tissue is in excess, the products of earlier decay being more rapidly absorbed, and leaving only slight traces here and there, the two processes going on side by side, and no actual tumour production occurring.

But even where gummata of some size exist some infiltration is also found, and in many cases the gummata gradually become more or less absorbed by the vessels of the fibrous bands running into their interior. I am convinced that there is no distinction between the elementary composition of infiltrating syphilides and that of gummata, and that we ought to speak simply of syphilitic new growth whatever its form.

In its later periods the growth becomes gradually converted into a more or less complete fibrous cicatrix, retaining, however, for a very long period traces of its cellular origin, and always the high degree of vascularity and the peculiar arrangement of its vessels due to their mode of formation. From a comparison of syphilitic cicatrices from various organs with other scar tissue, I am inclined to believe that the microscopic characters are in nearly all cases sufficient for their distinction.

I shall not endeavour to show the bearings of these points on the nature of the disease, nor to inquire what light is thrown upon it by the kind of tissue implicated, questions which will be better left until further observation and discussion with the aid of specimens are possible; but I would point out that the vascular and lymphatic systems seem to be especially involved. I have not entered upon the subject of affection of non-vascular tissues.

If it can be shown, as I believe it may, that the syphilitic new growth, in whatever form, is of the same ultimate composition, and follows the same course of growth; if, too, the existence of this growth is observed in cases dying at all periods of the disease, it seems to follow that it must be due to the continuous operation of one and the same cause throughout. And if it be allowed that this cause is the action of the syphilitic poison in the blood at one time, it must also be allowed to be so at another. The process is, to a certain extent, peculiar and specific, and proves peculiarity or specificity of the cause the more clearly because it is the process of growth, and not the tissue itself which constitutes the peculiarity of the product. And this, if true, would tend to prove that whether the morbid condition

be a modification of the blood or plasma, or an actual poison circulating in it, so long as any syphilitic growth is going on, the disease cannot be considered as terminated. Hence it is difficult for me to understand how gummata can be regarded by Mr. Hutchinson as *sequelæ*, unless the word is used merely in the sense of *results*. We are, I think, justified in assuming that the tertiary so-called gummata, as well as the infiltration, are simply growths, as Mr. Simon has said, which are due to the uniform action of the same cause, *i. e.* so long as there is syphilitic poison it goes on producing syphilitic growth.

There can be now no question as to the occurrence of actual gummata during the secondary stage, both in acquired and in infantile syphilis, the evidence being abundantly conclusive. I need only refer to Virchow and Wilks as bearing testimony to this fact. But infiltrating growths will, no doubt, be found with greater frequency at this period. It is so difficult to decide the limits of these arbitrary "stages," that it may often be a question as to which is to be selected for a given case. I have only had one opportunity of examining the body of a person who presented secondary lesions. It was that of a female, twenty-one years of age, who died from heart disease. There was a sore near the margin of the vulva, and some ulceration of the vagina; the tonsils presented recent extensive ulceration, and there was slight superficial ulceration of the pharynx. On the inner surface of the dura mater over the convexity of the hemispheres were a few scattered minute nodules, presenting all the appearances of minute gummata. Unfortunately the brain was not examined microscopically. The precise date of infection could not be ascertained, but it was probably less than eighteen months.

If I may venture now to turn to the subject of the general pathology of the disease, I would suggest that the analogy of syphilis with the zymotic or acute specific fevers has been somewhat overstrained. Up to a certain point a very close analogy must be admitted, especially when the latter are inoculated. In both there is an inoculation or entrance of the poison at some point in the system, as *e. g.* in smallpox or vaccinia. Then follows a period of elaboration and development of the poison, probably either in the tissues of the part or in the lymphatic glands, constituting the incubation period. Then follows the sudden diffusion of the poison into the blood, probably either in the form of solid particles or organised elements derived from the lymphatic system, or of a poisonous fluid; this gives rise to a fever, the onset of which may be

marked by rigors, and which constitutes the exanthematous fever which occurs also in syphilis, though usually in a slighter degree; we have, too, within a short period the occurrence of a rash. From recent researches it seems probable that all these symptoms may be due to the diffusion of germs or particles into the blood, which give rise by embolism to the symptoms of fever, and also to the proliferations of tissues and minute congestions or extravasations, and the like, which we call "rashes" of various kinds. The symmetry of the rash seems to depend, as Dr. Moxon has put it, on the fact that the blood is an impartial carrier to all the organs and tissues. Thus far the analogy of syphilis with inoculated specific fevers is complete. But here the analogy appears to me to cease, and the true blood affection to begin. In the acute specific fevers the morbid process has reached its maximum at this point; for a short period other changes go on, due in part to the progress of the lesions thus set in action; but speedily the poison germ is either eliminated or becomes effete; after having multiplied or "seeded" at the expense of the organism, it is extruded, and nothing remains but its effects, which are "*specific*" only in a limited sense, that is to say, the weakness or inflammation tends to affect certain organs or tissues, but no specific or peculiar product is produced. With syphilis the case is different; from the period of the diffusion of the poison into the blood, changes occur in the blood and tissues of a peculiar and specific type, going on perhaps for years, and affecting the whole organism in a special manner. The analogy seems then to fail from this point, and we may fairly ask whether the early resemblance is not due merely to the similarity in the mode of entrance of the poison; in the one case the poison being as it were an epiphyte, which grows at the expense of the system, and is then (itself or its offspring) ejected; in the other the poison depending on a modification of the tissue elements themselves, and growing from them, is capable of indefinite multiplication, modifying the whole system, and bending it to its laws of growth, just as the elements of a morbid growth tend to produce their like by the infection of healthy tissues. Nor is there any real bar to the acceptance of this view in the fact that the contagiousness of syphilis seems to be limited to certain products of the secondary period. We need not seek far in vegetable physiology for cases in which there is a power of reproduction by seeding only at certain periods, and the plant can also be reproduced by grafting or budding at any time. The chancre and certain secondary secretions are, so to speak, the

fructification and seeding of the morbid process, but there is abundant evidence that the disease may be afterwards grafted or budded in other ways.

From this time, then, it seems to me that we must regard syphilis as truly a *blood* disease, modifying probably the blood-cells, and through them the nutritive fluids. From this period we have a tendency to the production throughout the system of growths which follow a certain definite order and type, either as minute growths, infiltrations, or larger tumours, forming the class of symptoms known as tertiary.

In addition to these, however, the long course of infection of the blood gives rise to certain changes in the nutritive properties of that fluid, which may lead to the production of common irritative or inflammatory changes (such as are also caused by the poison of rheumatic fever and gout); hence probably arise some of the pains in the joints, affections of the serous membranes, and of the lining membranes of the heart and great vessels, such as peritonitis, endocarditis, &c. To similar causes it is highly probable that we must refer lardaceous degeneration of the viscera.

But beyond all these, in the long course of the affection of the lymphatic system and the blood there appears to be produced a degeneration of the nutritive processes, analogous to that observed in some so-called "constitutional" diseases, and which displays itself mainly by the imperfect nutrition and re-formation of certain organs or tissues. The changes of which I speak are more commonly seen in those cases where the disease has lasted for a long time. There are certain lesions almost peculiar to so-called hereditary syphilis which seem to be of this nature, such as the affection of the bones, the teeth, and the cornea. Some of these may occur at a long period after the occurrence of the true syphilitic symptoms. But I have been led to doubt whether some of the symptoms of so-called "hereditary" syphilis are not merely due to an heredity of a specific form of malnutrition or tendency to early decay of peculiar character, and not to the existence of actual syphilis in the offspring.

In the syphilitic parent we find that there is a great over-production and mal-production of connective tissue. Now, if Mr. Hutchinson's view be correct, viz., that syphilis is a specific fever, it would be an additional proof of that view, if the inherited portion of it were not the disease itself but something merely the *effect* of that disease. And may we not readily believe that this excessive malformation

of connective tissue in the parent may be exhibited in the offspring by a tendency to early degeneration or malformation of certain of the more highly developed or specialised forms of connective tissue, such as the cornea, teeth and bones; and that they are an expression of the exhaustion of the power of connective tissue formation.

I should like to ask Mr. Hutchinson whether there is evidence that in *all* cases of so-called hereditary syphilis—that is, the cases which present only notched and pegged teeth or interstitial keratitis—the children have always suffered from symptoms of true syphilis in infancy.

It may be urged that syphilis may exist during infancy, and that its existence may pass unnoticed, or a slight rash may be the only thing which is observed and this be ascribed to something else, or that where no history is attainable syphilis must be supposed to have existed. Now, if due to actual syphilis in the child itself, these lesions should be seen in cases of early vaccinal syphilis, or should be more likely to occur in them. Is this the case? The fact that a child grows to resemble its parent much more towards the time when these lesions show themselves must also be remembered. Again, a peculiar form of hyperostosis, usually considered peculiar to infantile syphilis, is in rare cases met with or closely simulated in very long standing-cases of acquired syphilis. This form of bone disease has been studied by many observers, but more especially by Valleix formerly and Parrot of late years.[1] This disease, common in hereditary syphilis, occurs in rare cases in long-standing acquired syphilis. I hope shortly to bring before the notice of the Society the case of a man who had been the subject of progressive syphilis for twenty-five years, and who presented the most advanced degree of syphilitic hepatitis I have ever seen. In this case there was a condition of the long bones precisely similar in all its characters to that described as peculiar to infantile syphilis, allowing of course for the differences between a developed and a growing bone. The condition too was such as I have never seen in any other form of bone disease. Now, this change in the bones, so slowly produced in this case occurs, from the earliest date in the child, and may not improbably be merely due to an inherited tendency showing itself in their development.

[1] For a concise description of the changes found in the bones in the several stages of the affection, I may refer to a recent paper by M. Parrot, published since the above remarks were made, in the 'Archives de Physiologie Normale et Pathologique,' No. 2, 1876, p. 133.

The fact that actual symptoms of syphilis exist in some of these cases does not at all negative this hypothesis. The child under such circumstances may and probably will have both heredity of peculiar malnutrition *and* communicated syphilis, and the interstitial keratitis or pegged and notched teeth may yet be due to the former alone. As presenting some analogy, and showing how a general constitutional influence acting on the parent during gestation may affect the development of the child *after* birth, I may mention what occurs in cretinism. It is well known that goîtrous parents living in a locality where goître is endemic may during their residence in that locality beget children who become cretins, but if they remove from the locality the children subsequently procreated do not become cretins. Now it must be remembered that cretinism is a condition which develops after birth, and that it depends, according to Virchow, on an imperfect development and early ossification of certain portions of the base of the skull.

Hence, whilst I do not for a moment question the connection of these lesions with heredity from a syphilitic parent or parents, I think the above hypothesis tenable to explain their mode of production. Moreover, many cases are known in which these persons acquire syphilis; this, though not a proof, is an *à priori* argument that they have not had true syphilis.

And here I must remark that I am led to question whether infantile syphilis is really *hereditary* in the strict sense of the term. There seem to me to be some weak points in the argument. It is said—well, there are cases on record, in which careful minute investigation was made, and no symptom of syphilis, past or present, was to be discovered in the mother. But as I have said, and Mr. Hutchinson would I think allow, this is no proof that the mother has not had it. Cases are frequent where syphilis shows itself by tertiary symptoms long after infection, where there is no proof of secondaries having occurred; and of gummata being found in the body after death where no symptoms were observed during life. In the cases of one single coition, of which I believe some are on record, the evidence again is imperfect, and it is scarcely credible that frequent connections should occur during a long course of time, and the mother remain uninfected, and yet the child be infected by perhaps a single spermatozoon.

The period at which the specific rash and other symptoms usually show themselves in the child is very nearly the same as that at which

they occur after inoculation in the adult, and the disease may not improbably be communicated from the parent at the time of separation of the placenta, as has been suggested by Dr. Cory, when for a time there is possibility of direct communication of the blood of mother and child, or at birth. But on the other hand there is the fact that, as Mr. Hutchinson has said, the poison seems to lie dormant in some cases. As an illustration of this I may mention a case now under my care.

A child was vaccinated at the age of $3\frac{1}{2}$ months; previously to vaccination the skin was perfectly clear and free from eruption. At about six or seven weeks from vaccination a "measly" rash, as the mother called it, suddenly appeared, and increasing spread all over the body. Vaccination was performed on January 1st, the child having been born on September 12th. I saw the child on March 8th; it was then covered with a rash, mainly of papular character, large coppery blotches, some large ulcerated papules, &c.; it was, in fact, a typical case of syphilitic eruption. Since then some mucous tubercles have developed.

The vaccination marks were very distinct, one scab not yet being separated. But there was no induration whatever of the scars, and although the glands in the right axilla, on which arm the vaccination was done, were somewhat enlarged, they were equally enlarged and indurated in the left axilla. Moreover, on further inquiry I found that the mother had had three miscarriages at a period of about six months, and that the child had snuffled from the birth. Hence it seemed more probable that this case, at first sight a typical case of vaccinal syphilis, was really one of congenital nature, and that the irritation of vaccination had merely served to evoke the dormant poison. I mention this case as being in some measure confirmatory of Mr. Hutchinson's views on the cause of appearance of the rash at the particular time when it is usually observed.

In conclusion, I cannot but express the hope that this discussion will serve to direct attention to some of these difficult points and to lead to their elucidation, and this must serve as my apology for bringing these considerations before the Society.

Dr. Septimus Gibbon.—With reference, sir, to the important remark of Mr. Simon, that he has witnessed a case of syphilis in the third generation, I wish he had specified the lesions observed, because I entertain serious doubts as to the truth of Mr. Hutchinson's doc-

trine that there is no relation between syphilis and tubercular disease. It has been my lot to meet with cases of tubercular diseases whose origin I could attribute to no other cause than syphilis in the paternal grandfather. They occurred in Hebrews, who are notoriously exempt from scrofulous and tuberculous taint. In confirmation of this fact I may, perhaps, be allowed to mention that during the last twenty-five years I have attended a large number of Hebrews, both as hospital and private patients, and have never met with a single case of pulmonary consumption amongst them, and a former colleague at the London Hospital, who had a large *clienitéle* amongst this favoured people, told me that for forty years he had only met with two cases. The medical officer of one of their large schools has remarked that their children do not die in anything like the ratio of Gentile children; and in the district of Whitechapel the Medical Officer of Health has reported that on the north side of the High Street, occupied by Jews, the average death-rate is 20 per 1000 inhabitants, whilst on the south side, occupied by English and Irish, it is 43 per 1000. Side by side with this superior vitality and marked exemption from scrofula we have the facts that their females do not practise prostitution, and that circumcision, as conclusively proved by Mr. Hutchinson, confers on their males an immunity from syphilis four times greater than that enjoyed by Christians. In these broad facts there appears to me to be some presumptive evidence that syphilis is the parent of scrofula in, it may be, remote generations.

When Jews have syphilis they suffer severely from primary and secondary symptoms, and little, if at all, from the tertiary. I can point to patients who are the pictures of health, and would, I fancy, defy Sir William Gull's nasal test, as they certainly do the anæsthesia test relied on by some French practitioners, and yet they have begotten syphilitic and unhealthy children.

For many years I have attended a family where the grandfather admitted that he had syphilis; his children are anæmic and constantly under treatment for dyspepsia, rheumatic pains, and slight ailments, but have had nothing of a tubercular form since I have known them. I am unable to say whether they exhibited the symptoms of inherited syphilis in infancy; they certainly show none of the physiognomical signs of the inherited disease. But the grandchildren in two families have had scrofula in various and unmistakable forms; one child has had scrofulous abscess of the testicle,

another psoas abscess, another scrofulous disease of the bones of the arm, and others ulcerations and abscesses about the neck and ears. All have contraction of the upper jaw. On investigating the family history of these cases I failed to detect any scrofula taint on either the father's or mother's side. Is it not, therefore, a fair inference, from such cases as these, to conclude that syphilis is *one* of the sources of scrofula? I readily admit that there are other causes, such as want of bodily exercise and residence in damp and dark places.

As to the classification of syphilis with the exanthematous fevers, I cannot agree with Mr. Hutchinson, and, in addition to the numerous points that have been mentioned, I would point out that the analogy does not hold good in the matter of treatment. Mercury modifies and controls the symptoms and duration of syphilis; some surgeons even hold that it is a specific cure for the disease, and I would ask Mr. Hutchinson whether there is any specific or any agent that will modify or control the symptoms of measles, smallpox, or scarlet fever? Did he ever see these diseases or any exanthem either cured, cut short, or modified by treatment?

Although mercury and iodide of potash temporarily remove the blotches, the iritis, and "gummata" of syphilis, I do not regard them as specifics, because in the great majority of cases when the remedy is left off these symptoms return, and often the last state of the patient is worse than the first; certainly, the worst cases of rupial sores, nodes, and necroses of bone I have witnessed have been in patients treated by mercury. I believe Mr. Hutchinson based his doctrine of mercury being a *specific* for syphilis on a series of vaccinal syphilis in children and young adults, because under its use the symptoms passed off; but, as Dr. Drysdale has truly observed, the symptoms in such cases would almost as readily have passed away of themselves without the use of mercury. I am inclined, sir, to think that we shall best discover an antidote, and get at the true nature of this wide-spread and fearful disease by a thorough and minute investigation of its action on the blood in the secondary stage. If the Society would undertake such a careful investigation it might, perhaps, decide whether it is a poison or a fermentative parasite, as well as its relation to the exanthemata and other points so ably dwelt upon in this discussion. The action of the poison is apparently to break up the red globules of the blood, and to allow the hæmoglobulin to escape and form blotches on the skin.

Hence, in contradistinction to the *medical*, it might be called a *surgical* exanthema. The best observers state that the red globules are not again restored in anything like their former number; the consequence is that the supply of oxygen to the tissues is very seriously diminished, and the effects on nutrition and ordinary health are most disastrous. Is not this large withdrawal of oxygen from the blood and tissues sufficient to explain the formation of nodes and gummata without the far-fetched hypothesis of Mr. Hutchinson and other speakers, that they are the direct product of the poison or regrowths of the parasite? I am disposed to think it is, for the reason that all nodes and "gummata," if treated moderately early by mercury, iodide of potash, or even peroxide of hydrogen, whose actions, so far as we know them, are simply to convey oxygen to the tissues, can be quickly removed, but if kept too long they are difficult of absorption and may induce other morbid changes.

Adjourned Discussion, April 4th.

The President.—Perhaps before I ask Mr. Hutchinson to reply to the various observations that have been made, you will allow me to make a few observations which I would have done at our last meeting, had it not been that we were so late in our discussion, and I was unwilling to detain you. I hope Mr. Hutchinson, being present, will accept anything I say in a friendly spirit if we should differ, for I confess I have felt very much like a pupil sitting at the feet of a teacher in listening to all I have done from Mr. Hutchinson and others, having learnt much and being still anxious to learn more. Mr. Hutchinson says, "Are there any facts which favour the belief that syphilis continues to be a blood-disease, after the cessation of all tendency to produce symmetrical symptoms?" I think the discussion that has taken place has produced sufficient evidence to show that we are not agreed upon this point. When we have to deal with such a poison as syphilis, I think we must look at it entirely as a disease *per se*, as stated by Dr. Wilks, to whose observations, I think, everybody must have listened with very great interest and instruction. If we contrast syphilis with the various eruptive diseases attended by fever, we find that each of the latter runs its limited course, terminates at a stated period

without leaving any consequences, and is not influenced by any medicine or treatment. The disease terminates without leaving any sequelæ of a specific character, and to this I shall refer presently; but it leaves the patient in a condition which usually renders him free from any subsequent attack. As a general rule, he has no power of infecting his offspring with that disease, nor does he render that offspring free from its contagion conveyed to it by any other means. But with regard to syphilis, we see a very small poison introduced into the system, followed by an eruption, and followed by those consequences which are termed the sequelæ of that eruption—the tertiary condition. I agree with Dr. Wilks in thinking that the terms "primary, secondary, tertiary, and quaternary" really are of very little importance; they rather map out phases of the disease, than indicate anything very specific: and when we see the disease in its secondary condition evidenced merely by an eruption, we really should consider that that eruption is only one indication of a very large poison affecting the whole system; and how far we can take that secondary eruption as an evidence of a blood-disease, and that that blood-disease terminates when the eruption is no more symmetrical, but, disappearing, leaves behind it certain conditions which we term tertiary symptoms, does not appear to me at all clear. Mr. Hutchinson has stated that it would be rash to assert that it was impossible for tertiary syphilis to prove contagious, but at present we have no evidence which would support this affirmation. With regard to that, I should like very much to ask Mr. Hutchinson if he is satisfied that we should not get infection from inoculation of a healthy subject from a tertiary sore or tertiary discharge or matter from any person affected by tertiary syphilis; and unless we had these experiments largely carried out, I think we cannot safely arrive at any satisfactory conclusion. Mr. Hutchinson says the risk of contagion appears to cease long before the risk of hereditary transmission. I should like to ask him if he can give us any hint as to how he arrives at this conclusion, and if he can tell us when this tertiary condition ceases to be innocuous, and enables the father to beget a healthy child. That a person suffering from tertiary syphilis is capable of producing disease in his offspring we know to occur; and I mention the following case as illustrative of a point which I propose to follow out in asking Mr. Hutchinson a question. A patient was admitted into

St. George's Hospital with a syphilitic affection of the larynx. She was suddenly seized with great difficulty of breathing and impending suffocation, and I had to perform tracheotomy upon her at a moment's notice. The evening of the operation, she was delivered of an apparently healthy child. The next day the nurse drew my attention to a sore on the back of her left thigh, and that was a solitary unsymmetrical rupial sore; so that, taking Mr. Hutchinson's description of the conditions of rupia, that when non-symmetrical it is of the tertiary form, there we had evidence of the tertiary condition of syphilis giving rise to what within three weeks proved to be a syphilised child. The child within three weeks, although born healthy, was covered by an eruption about the nates, scrotum, and the lower part of the body. To carry on this point with regard to the condition of the blood-disease in the primary and the secondary or the tertiary stage, I wish to mention another case. A woman was admitted into the hospital with a hard smooth ulcer on the surface of the left breast. The base of the ulcer was extremely hard, and that went on gradually increasing slightly in size, and was attended by a slight copper-coloured eruption. I particularly went into her history, and it appeared that the woman had lost her own child, and had had another child sent to her to be nursed; that child was covered by a secondary eruption, with a sore on its mouth, and had given rise to a chancre of an undoubted character on the nipple of this healthy woman; and under a course of mercury she ultimately recovered. If, in the tertiary condition of syphilis, accepting Mr. Hutchinson's theory or interpretation, we have the disease ceasing to be a blood-disease, and becoming merely localised in the tissues, soft or hard, I ask how he explains that the tertiary disease gives rise to the secondary in the offspring, and the offspring that has secondary disease produces the primary sore on the breast of a healthy woman? When is it, or how is it, that the tertiary disease ceases to be a blood-disease, and how is it that it merges by transmission into the primary condition of a blood-disease? Then if the tertiary disease be not a blood-disease, but a local disease—I speak as a surgeon—I would ask him how it is that in plastic operations, attempted upon a patient suffering from tertiary syphilis, we do not get union—that the wound is affected by the condition of the patient? A medical gentleman had been suffering from acute syphilis with gonorrhœa; he had an abscess in his perinæum, which was rather boldly opened, and unfortunately a communication made between

the bladder and the rectum. He came to me two or three years afterwards with tertiary affection of the nose and the palate, in the greatest distress; he had no command over the bladder, and begged me to operate upon him. Seeing his condition, and seeing how saturated he still was with the poison of syphilis, I begged him not to undergo an operation, for I was certain it would fail. However, he pressed me so hard that I at last consented, very much against my own judgment; and operated upon him. He had considerable secondary hæmorrhage, and there was not the slightest attempt at union. I would ask any surgeon present if, with tertiary syphilis affecting the face and nose, he would not allow a considerable time to pass before he would attempt anything like a plastic operation, where union by first intention is so necessary for success? These are the points that have occurred to me, as militating against the idea that you can separate the secondary from the tertiary conditions of syphilis. Or rather, is it not more important that we should consider the disease as a whole, and in its tertiary condition that it is running on perhaps to its end, mitigated by treatment, moderated by time, altered in circumstances by those daily changes that take place in all structures, and perhaps approaching that end, which by a little more treatment and by patience may, at any rate, place the patient in a condition in which he may be considered free? But experience teaches us that, under adverse circumstances, mental distress, exposure, depression from treatment necessary for other illnesses, the disease may manifest itself again, and at any period of life. Many years ago, when in Canada, I was asked to see an officer between fifty and sixty years of age. He had two or three soft nodes on his skull; he had been treated for a severe attack of bronchitis in the middle of a Canadian winter. In questioning him whether he had ever had syphilis, he seemed rather surprised, and reverted at once to his ensigncy in New South Wales, where he had contracted syphilis; he had almost forgotten the circumstance. There must have been an interval of nearly forty years between his primary sore and its tertiary development; and I looked upon that as the development of the tertiary symptoms the result of his deteriorated condition of health and the treatment which he had undergone. These are the points which appear to me to bear very strongly upon the fact that, as long as tertiary syphilis manifests itself, we should consider it syphilis, to be treated as syphilis under any other circumstances; or, as Sir James Paget said,

with iodide of potassium for its passing cure, and mercury for its ultimate relief and eradication. With regard to the point that Mr. Hutchinson brought before us—and, I think, with very great ability—namely, how far syphilis is connected with scrofula, or how far it produces scrofula, I believe that there is no evidence that one can satisfactorily rely upon that syphilis is in any way the parent or the relation of scrofula; but I believe scrofula may very materially influence the severity of the syphilitic attack, just as other circumstances may do. I think this is a point of very considerable importance: we have climate, we have constitution, we have temperament, we have also the nature of the poison, to a certain extent, influencing the condition of the patient who is running through a course of syphilis. I have an interesting communication from Dr. Duka, who has seen a good deal of syphilis in India; he says, with reference to the manifestations of syphilis in India, "it seems that the virus of mixed races—that is to say, a European infected by a native woman—produces a more obstinate disease; the primary sore also being more liable to run into the phagedænic type than is the case otherwise. There is no doubt, however, that the natives are much disposed to the ravages of the disease." As far as my experience goes—and I think very likely Mr. Hutchinson will confirm this—some of the most severe cases of tertiary syphilis I have seen have been the result of disease contracted in China and in India. I think it is necessary that I should refer to what Sir James Paget said with regard to the sequelæ of syphilis in any degree resembling the sequelæ of other fevers. Now, I cannot bring myself to agree with Sir James Paget in this, although I hope in most matters we should agree. I look upon the sequelæ of fevers—and I think I shall be supported in this view by many—very much as accidental. They are in no degree related to the fever itself, after whatever type of fever they may follow. Take the sequelæ of typhoid: I have generally considered the sequelæ of typhoid as a pyæmic condition rather than as anything due to the typhoid-poison; and I have seen these sequelæ symmetrical as well as non-symmetrical, two nodes occurring upon the tibia in more than one instance. And I think those sequelæ resemble in their character the sequelæ of lying-in women—phlegmasia dolens; or phlebitis observed in cases of foul abscesses in the lungs. Therefore, I do not think there is any analogy between the sequelæ of syphilis and the sequelæ of other fevers. It is a pity that we should confound

the two. I look upon the sequelæ of syphilis as syphilis, and upon the sequelæ of other fevers as purely accidental. I have ventured to introduce these few points as they have occurred to me; but I cannot sit down without congratulating the Society on the very able paper that we have received from Mr. Hutchinson, and congratulating Mr. Hutchinson upon the very fine chain of philosophical thought that runs through the whole of his communication. I think the Society is very much indebted to him, not only for the paper, but for the discussion that has followed it. I received a letter from a friend who was present at one or two meetings, and who has read the other portion of the discussion; he writes to say the only conclusion he could come to was that surgeons, as well as doctors, differ. Well, if we differ, gentlemen, in one or two points, it is merely a theoretical difference, I believe; and a difference which perhaps will not prove of any serious importance; for, after all, this question whether syphilis in its primary condition be a blood-disease, and in its tertiary condition is a disease of the tissues, is of no importance to us. We shall not differ with regard to the true pathology of syphilis, for the knowledge of which we owe so much to Dr. Wilks and others; nor shall we differ very much with regard to the treatment. Perhaps it is a little diverging from the point if I say here—and I think it will be acceptable to many of us—I do not think we shall differ in the view that perhaps this discussion may strengthen the hands of those who are moving for legislative enactment to suppress to a certain extent the evil of syphilis; or, at any rate, to modify the great amount of syphilis that exists amongst the lower classes. In saying this much, I will conclude merely with a hope that the light which was spoken of by Sir James Paget may, by the work of Hutchinson and others who have laboured so largely in the field of syphilis, be yet held aloft as a beacon to guide us, and steer us straight in the troubled sea of the complications of syphilis.

Mr. Jonathan Hutchinson.—I must begin first with an apology to you, Sir, and to the Society, that I was not present at the last two meetings; and I must say that it was from no intention on my own part, but from circumstances quite unavoidable. I have, however, very carefully studied the very accurate reports of the discussions in the journals, and I hope I shall not do any speaker

injustice from not having been present. I feel also that I have reason to congratulate the Society on the fact that my absence on the last occasion was the means of procuring for the Society the speeches of Sir William Gull, of Mr. Simon, and of Dr. Greenfield, all of which we should have been very sorry to have missed. I think I perhaps also ought to apologise for the fact that I am going to read my reply; but it is long, and I am sure, if I did not read it, I should occupy very much more of the Society's time, and probably to less purpose. With regard to the remarks which have just fallen from yourself, I am exceedingly obliged for them, because they have given me a sort of summary of the discussion, and expressed very concisely your views, and I dare say the views of a great many, as regards the chief points which have been raised. I am further very glad to say that I find you and I differ scarcely at all; it is only on the interpretation of one or two minor facts. As regards most of what you have said, I have dealt with that in the reply I am about to read. With reference to your remarks as to the inference from the fact that a surgeon would not like to operate upon a patient who was the subject of tertiary syphilis, believing that the wound would not heal—

The PRESIDENT.—A plastic operation.

Mr. HUTCHINSON.—Admitting the fact to the full extent you have stated, I should not allow that it implies any reason for believing that the blood was tainted.

It will, sir, I think, have been obvious to most that two principal ideas ran through the remarks which I had the honour of addressing to the Society in introducing this discussion. The first and most important was, that syphilis depends upon the introduction into the system of a living material which is capable of self-multiplication, which breeds in the blood and tissues, and which is destined to pass through various stages of development, and finally to die. My second idea was, that, in order to a correct clinical comprehension of all that follows on syphilitic poisoning, we must admit, in connection with the phenomena which rank as its direct results, certain less closely associated peculiarities of inflammation and of cell-growth. In this way, I tried to show that soft chancres might result from contagion with pus produced by syphilitic inflammation, but not actually containing the syphilitic sporules; that phagedæna in like manner might produce a contagious secretion, but not a syphilitic one, and that it might wholly escape the restraint

of the specifics for syphilis, and that a gumma might grow long after a patient had ceased to possess either in his blood or tissues the living virus of his original malady. These various events were, I suggested, to be reckoned rather as adventitious to the syphilitic fever than as true parts of it; and to the later forms of gumma, &c., known as tertiary symptoms, I thought the name *sequelæ* the most appropriate, meaning by that term that they came after the true syphilis was over, just as we know that as a sequel to war may come a famine, though the war itself is ended. It will, I think, be readily granted that the correct appreciation of the relationship of the soft sore, of phagedæna, and of tertiary symptoms, to syphilis itself is all important as regards our insight into the kinship of the latter to other more short-lived exanthems. If the tertiary symptoms are not, in a strict sense, a part of syphilis, and if in reality the latter be usually over in a year or two from its outset, we are able at once to realise much more easily the kinship referred to.

Now, in looking back on the very important criticisms which have fallen from the various speakers who have so ably sustained the debate which ends to-night, I find, on the whole, a very considerable amount of concurrence in the views expressed; by no means, however, without some expressions of misgiving and dissent. By one or two it has been hinted that there is no proof of the existence of a germinal poison as the cause of syphilis; others have thought that the analogy with specific fevers has been pushed too far; that the stages of syphilis are so irregular, that they can scarcely count as such at all, and that it is wiser to speak of the disease as one whole; whilst many have expressed doubts as to whether it be possible for any results to persist, unless the blood still continue tainted. To take the first of these objections, I have to reply to Mr. de Méric—who reminded me that the germ-poison of syphilis had never been put under the microscope—that surely there are cases where deduction amounts almost to proof. With regret I admit that I have never seen with my outward eye the cryptogamic germ-poison of syphilis; but to my mind's eye it is as certainly present as if I had. Some one will see it some day, for it is beyond doubt that it must be there. I would even venture to suggest that it might be wise to anticipate discovery a little further, and to speak of this *contagium vivum* as the *syphilitic yeast*, so that we may force our minds to keep clearly in view the possible developments

of this theory both as regards the inherited and the acquired disease. When a better theory is forthcoming, we may lay this aside, together with the forms of phraseology which it has brought in; but I cannot but note, as a remarkable result of this discussion, that, despite certain objections to this, there seems to be no other theory in the field. For the present, to abandon the germ or yeast hypothesis of syphilis would be to throw the subject back into confusion; for it, as far as I can see, is the only clue which we possess to the orderly arrangement of our facts. Whether or not it will, when carefully developed in detail, be proved sufficient to explain all the facts, is the problem which confronts investigators. For myself, I may admit that the result of this debate has been to strengthen my belief that it is not probable that it will ever be supplanted. I have failed to see what those who suggest that the analogy with the exanthems has been pushed too far, have advanced in support of their criticism. I can only again express my conviction that the more the facts as to syphilis are examined, the more clearly will it appear that, when allowed to develope without interference by specifics, its stages are very fairly regular, whilst the more we know of the other exanthemata, the more willingly shall we admit that theirs are by no means so definite and precise as we are accustomed to assume. Whether or not it results from our habit of prescribing remedies from which no very definite effects are expected, I cannot tell; but certainly it seems wonderfully difficult to persuade ourselves that mercury really does interfere with the development of syphilis, and that it is hopeless to attempt to get correct ideas of the natural course of the malady, if we investigate cases in which it has been used. Amongst the facts which chiefly impressed on my mind the lesson that syphilis, when let alone, is really a very orderly disease were the two now well-known series of cases in which, some years ago, I had the opportunity of examining a number of patients who had all at certain definite dates been made syphilitic by vaccination. In the first of these, about ten persons had indurated chancres on their arms at the same time, and due to the same cause. I do not think that any two of their sores varied more than a week in stage of progress from each other. The similarity in date of induration, &c., was most remarkable, and quite as definite as would have been the phenomena of vaccination or of smallpox in a like series of cases. In my second series, the stage of the disease was further advanced before the accident was dis-

covered; and here, again, we found syphilis very regular, since case after case was sought out and found to be in almost exactly the same stage as its fellows.

If, however, mercury be given, the course of things is wholly altered, and it depends upon circumstances whether the exanthem stage be wholly prevented or only very much deferred. In a case in which mercury was begun early and continued for six months, I have known the rash, which would otherwise have come within six weeks, deferred till the end of the period named. Here I am obliged to interpolate that I did not exactly see Dr. Farquharson's meaning, when he said that mercury makes syphilis more regular. That it makes it milder is undoubted, and that it makes its stages, as a rule, much shorter is most undoubted; but I feel sure that, in certain cases in which it fails to cure, it may greatly protract the malady by increasing the distances between its stages. The theoretical explanation of this is, that it probably prevents the development of the yeast in the blood; and, if it do not wholly kill, it leaves it still able at some future time, when the antidote has been laid aside, to resume its growth. In connection with this theory, I wish to direct attention to a most important suggestion which fell from Dr. Broadbent, and which was especially welcome as a striking exception to the remark I have just made as to the general incredulity now prevalent as to positive therapeutics. Dr. Broadbent remarked that, now that it has been proved that mercury is an antidote for syphilis, and made probable that the syphilitic virus is the same in its nature as those which cause the exanthems, we ought to again make careful trials of that and other specifics for them. The same thought has often occurred to my own mind. If mercury can kill or retard the development of the yeast-plant of syphilis, it is very probable that it or the iodide, or some similar remedy, may do the same for the yeast of typhus, typhoid, or small-pox. We must not assume that this question has been set at rest by any trials which have as yet been made; for they have not, I think, been carried out with sufficient care. It will be desirable to ascertain carefully, beginning at the very earliest possible period, whether the evolution of the disease, as tested by the temperature and other symptoms, can be modified; whether the stages can be protracted; and, this point having been set at rest, we must then inquire whether such retardation is, on the whole, for the good of the patient, and determine the clinical details to which it may be

necessary to attend. It is quite possible that the same remedy may be very useful in one mode of administration and very hurtful in another, and thus the statistics *en gros* of recoveries and deaths under mercurial treatment in typhoid, which have been collected hitherto, may have no close application.

The discussion which has taken place as to when syphilis ceases to be a blood-disease has been very important; but it has been in part based upon a misapprehension, and it has elicited expressions of opinions rather than facts. There has been, I think, a general tendency to hold that it is probable that, so long as any manifestations whatever exist, there must be a blood-taint. Several speakers have seemed rather to deprecate the attempt to mark out stages at all. Thus, one to whom the subject is very greatly indebted — Dr. Wilks — said that for him, when a patient had syphilis, he had syphilis; and another of high position in the profession informed us, pithily but apparently without regard to stage, that syphilis was not so much a "blood-disease" as a "flesh-and-blood disease."

Now, in reply to those criticisms, I cannot but still think that it is consistent with fact to divide syphilis into stages; and that the degree in which the blood and tissues are relatively affected by it differs very much at the different periods. No one will probably deny that syphilis, whilst breeding in the early periods of chancre is a tissue-disease only, its yeast is not yet free in the blood; then follows a period when the blood is its home, as may easily be proved by inoculation experiment. The blood cannot be infected many days—probably not many hours—before the poison finds its way into the tissues; and the misapprehension to which I have just adverted consists in this, that some speakers have seemed to suppose that by blood-disease was meant one in which the blood alone shares. I really cannot admit that the terms I used afforded any ground for such misapprehension; for the very pith of my argument as regards the symmetry of the phenomena in the secondary stage was that the tissues became infected by the germ-carrying blood. Of course, in its secondary or exanthem stage, syphilis is a flesh-and-blood disease, or rather, to denote sequence accurately, a blood-and-flesh disease. But, in the primary stage, it was local; and it is very possible that in the tertiary stage it may be local again. In other words, the poison or yeast may have died out of the rapidly changing blood; whilst it, or perhaps its results, are still present

in the solid and less mutable tissues. To determine this, we want not opinions, but facts.

Now, there are several kinds of facts by which the state of the blood in syphilis may be estimated. First, we have its contagious properties, or inoculability. This is conclusive; and it is quite certain that, during the secondary stage, the blood is contagious. The numerous accidents in vaccination and otherwise which have occurred during this stage have fully proved, both for the acquired and inherited forms, that the blood may be rich in contagious material, even when the patient displays no external symptoms. Although, as a rule, when the blood contains the poison it will produce an eruption, it by no means invariably does so; and it may be admitted as highly probable that the blood continues to be contagious for a certain period after the external phenomena have ceased. How long is that period, and within what limits may its duration vary? That is the question before us. Most unfortunately we are precluded from experiment; for syphilis is with great difficulty communicable to the lower animals, and it is not easy to find conditions under which, in the human subject, such procedures would be justifiable.

Availing ourselves of such facts as accident throws in our way, I believe we can produce but little evidence in favour of prolonged contagiousness of the blood. All the accidents occur during the year or eighteen months which we count as the secondary stage, and most of them in the early part of it. There is every reason to believe that, in the tertiary stage, neither the blood, nor even inflammatory secretions produced by sores which still bear the specific type, can reproduce the disease. Our next test is the possibility of transmission to offspring; and I note that almost every speaker has been inclined to assert that the production of a tainted child must be regarded as proof of blood-poisoning still extant in the parent. This may be so; but I cannot help the conjecture that it may be possible for the germs to still hold possession of cell-structures in the ovary or testis when they no longer exist free in the blood. That such is the case cannot be proved, and must rest for the present as mere conjecture. It becomes, then, of great importance to answer the question—*How long after the secondary stage is it possible for syphilis to be transmitted hereditarily?* During the last few weeks, I have gone through the notes of a great many cases, in order to get data for a safe reply on this head, and

with the result of a strong impression that we have much exaggerated our estimates of the time. The cases in which syphilis is transmissible by inheritance for more than a year or two after its secondary stage appear to be very exceptional. I possess notes of a few cases in which successive children, during a period of seven or even ten years, have presented evidence of taint; but the ordinary course certainly seems to be that the first two or three children suffer, and that the others escape. Unless the risk of hereditary transmission did really cease early, in the vast majority of instances, infantile syphilis would be far more common than it is. It will be readily seen that our decision on this point is not a mere matter of speculation or of transcendental pathology, for upon it must rest the advice which we give our patients in reference to marriage. Now, I have for long made it a rule when consulted on this point to insist that, before marriage, a period of two years should elapse from the last of what I have considered blood-symptoms. I have given this opinion to a great many persons, and may confess that it has been a constant source of anxiety, lest some day some one should bring me a snuffling, spot-covered baby, and say: "See here; you said I might marry; just look at this!" Such an occurrence has, however, never yet happened to me. I may strengthen the bearing of this fact by adding that I have been cognisant of not a few cases in which the marriage took place at a much shorter interval than had been advised, and yet healthy children were produced. I repeat, then, that there is reason to believe that the instances of liability to transmit to offspring, extending over periods of several years are exceptional, and are by no means to be dealt with as if they illustrated the rule. There are, moreover, numerous fallacies to be carefully kept in mind in investigating cases of the supposed unusual prolongation of this risk. There is the almost certainty to which I shall have to allude directly, that the mother becomes contaminated by her fœtus, and thus, if healthy before, supplies a new starting-point for the infection of future children; and there is always the risk that one or other of the parents may have contracted the disease a second time.

Thus, then, we have clearly a period of syphilis during which the original sore is contagious; a period during which the blood and tissues are contagious; and a period during which transmission to offspring is possible. It is doubtful whether or not the two latter cease simultaneously, but there is some probability that the last

remains the longest; both, however, in almost all instances, end within comparatively short periods.

Let us ask, next, how it is with the symptoms which we rank as tertiary. Here we find a totally different law. The liability to them persists after the longest periods of apparent immunity, and after a whole family of healthy children have been produced. Nor are such cases rare; they are almost the rule. I am obliged, then, to again ask the question—Is it not far more probable that such symptoms result from changes in the solids which have taken place during the protracted secondary stage, than that they are consequent on a blood-taint still in activity.

At this point, the argument receives, I cannot but think, great support from the general fact that everything in the secondary stage is symmetrical, and everything in the tertiary unsymmetrical. The inference suggested from this is that the phenomena of the tertiary stage, not unfrequently single, are of local origin, and possibly not unfrequently acknowledge accidental causes; whilst those of the early are due directly to the circulation of poisoned blood. Now, I must confess that I have been a little astonished at some of the opinions which have been expressed during the debate on these facts as regards symmetry and the inferences from them. One speaker, if I did not misunderstand him, thought it an original statement that symmetry after all did not mean anything more than that the poisoned blood circulated equally on the two halves of the body. Now, so far from this explanation being novel, it is precisely that which gives the symptom its value. Others have alleged that, after all, secondary syphilis is not so very symmetrical, and Dr. Moxon has explained to us that, although it is symmetrical, yet its symmetry has no meaning, since it is invalidated by the "fallacy of universality." To those who doubt the fact of its usual symmetry, I have to say simply, strip your patients and inspect them in a good light; and, if this be done, I cannot conceive that there can be any doubt on the matter. Allowance must always be made for slight deviations from exact parallelism, for neither the spots on the wings of a butterfly nor the markings on a spider are ever absolutely symmetrical. It is recorded of a Dutch gardener that he caught a boy stealing apples, flogged him, and shut him up in one of his summer-houses; and that, having done this, his mind was so much disturbed by the interference with the symmetrical plan of his garden, that he could get no peace until he had flogged his own boy

and locked him up in the corresponding building. I was careful in my introductory remarks to say that we must not expect from Nature symmetry of the Dutch garden type. That in secondary syphilis the sameness in extent and location of the phenomena on the two halves of the body is such as ought to be quite conclusive as to its meaning, I still fearlessly assert. To Dr. Moxon, I reply that, if an eruption be so universal that you cannot judge of its symmetry, the fact of universality teaches pretty much the same lesson, and cannot often be explained on any other hypothesis than that of blood-contamination. Those, however, who are familiar with the syphilitic exanthem, know that it is not often universal, and that its symmetry is constantly seen in the most definite manner in cases in which it is only scanty. Sir William Jenner, directing his arguments in the same direction, has told us that the rash of typhoid fever, a blood-disease, is not symmetrical; whilst common psoriasis, the patches of which are usually symmetrical, is a skin-disease only, and may be produced by local causes. Now, there are few authorities to whom I would with greater pleasure yield my opinion, if I could, than to Sir William Jenner, but in this instance I am obliged to dissent somewhat from both his statements. If the rash of typhoid be really not symmetrical in any other sense than that it is often ill-characterised and difficult to see, it is a very extraordinary fact in pathology, and is well worthy the most attentive scrutiny of this Society. On the other hand, taking psoriasis as the very type of a symmetrical eruption, I must assert that it is probably not a mere skin-disease, and that it cannot be evoked by local causes. The influence of varying states of health and age, of lactation, &c., in producing it, and its cure by arsenic seem to prove that it is constitutional in origin; whilst if it were ever due to local causes, it could certainly never, under such circumstances, become symmetrical unless the irritation were equally applied to the corresponding parts.

Amongst those who have produced evidence in favour of the assertion that the phenomena of tertiary syphilis are, as a rule, non-symmetrical, my thanks are chiefly due to Dr. Moxon, whose facts derive additional value from the circumstance that he is an unwilling witness. Could anything be more conclusive as to the general fact on this subject than that a pathologist so able and zealous as Dr. Moxon, with Guy's Hospital as his field, mentions to us four cases of symmetrical tertiary syphilis, and four only. If the facts were as

he wished to imply, that no great difference in this respect could be established between secondary and tertiary symptoms, he would easily have been able to give us four hundred. I have myself repeatedly published cases of symmetrical tertiary syphilis, but the interest of the cases consisted in the fact that they were exceptions to the rule. It must be obvious that there is no law to prevent tissue-disease, such as I hold tertiary symptoms to be, from being symmetrical; on the contrary, the wonder is that they are not more often so, resulting, as they do, from long-standing disorder of nutrition which was caused at a time when the blood was tainted. So much the more significant is the clinical fact that, as a rule, they are not symmetrical. In repeating this assertion of their almost constant non-symmetry, I am speaking of the later tertiary phenomena, and chiefly of such as paralysis of the nerves of the eyeball or face, gummata in the tongue, muscles, or cellular tissue, and periosteal nodes.

Having mentioned Dr. Moxon's clever and amusing speech, it may be convenient here, perhaps, to conclude my reference to it, and to say that I am glad to find that he supports the exanthem doctrine of syphilis. I spoke of syphilis as a slow-staged or long-staged exanthem, and he calls it an "exanthem diluted by time." As to his suggestion that tertiary growths have their seat in portions of new matter, which, by some means, have got introduced into the system without parentage, and, therefore, without inheriting any degree of immunity, I shall prefer to avail myself of its author's permission to say nothing more about it, and shall await with interest its further development.

It has been prominently mentioned, as if it constituted a real difference between the exanthems and syphilis, that the former are not capable of transmission to offspring. The reply to this is, that such transmission is possible only during the period when the specific poison is living in the blood and tissues; and that this period in the more acute exanthems is very short, whilst the patient's condition during it is such as to utterly preclude the possibility of his becoming a parent. We cannot too clearly keep in mind this fact of extreme difference in length of stages, for by it are to be explained not only the possibility of hereditary transmission, but the subsequent liability to tertiary disease.

The statement made by a high authority, that syphilis differs from the other exanthems in having no pyrexia, I may conveniently answer

by quoting from Dr. Buzzard's speech at an earlier meeting. Dr. Buzzard, apparently as familiar with the earlier or surgical stages of syphilis as he is well known to be with those in which the nervous system suffers, spoke of fever as being "so significant a part." In point of fact, the pyrexia in the exanthem stage of syphilis sometimes runs high, and is probably always present more or less. I have known a patient almost die during it.

Amongst the important matters brought prominently under notice during this discussion, is the fact that there exists two schools of opinion as regards what takes place in the transmission of syphilis from parent to child. In the past, some, perhaps most of us, have held in a muddled sort of way both doctrines, and have allowed them to mix themselves up in a somewhat incongruous manner in our expressions and forms of belief. I cannot but regard it as a great gain that the two are now fairly confronted, since, without presuming to assert that they are incompatible, it seems very improbable that both are true. The one, which is the creed of those who think that syphilis is due to a sort of yeast germinal matter, holds that transmission is effected only by transference from parent to child of germs; that it is, in fact, contagion to the ovum. Thus, unless such infectious matter pass with the sperm or germ, no transmission of the disease will take place, and a child perfectly healthy, *quoad* syphilis, may be born. Transmission thus becomes to a certain extent a matter of chance, and by no means subject to the laws of ordinary heredity. The other view which, with the clearness and ability which characterise all that he teaches, was expounded by Sir William Jenner, makes the transference of syphilitic taint from parent to child but an example of heredity, in the same way that colour of hair, texture of skin, &c., are transmitted. "Potentiality of development, then, and not the state of the blood, not any appreciable change, is the real pathology, it seems to me, of infantile syphilis." I am quoting Sir William's exact words.

Let me try to state as briefly as I can a few of the facts in reference to these two doctrines. When syphilitic parents procreate, a not uncommon result is death of the ovum at very varying periods of intra-uterine life, a circumstance, probably, to be explained more often by disease of maternal structures (of the placenta) than by breeding of syphilitic virus in the infant. In another group of cases the infant is born apparently healthy, but sickens rapidly and, without any of the ordinary evidences of syphilis, but with pos-

sibly a pemphigoid eruption on the hands and feet, dies in spite of all treatment within a few days. It is very difficult to say what happens in these cases, or in what way death is caused; they are rare. Lastly, we have the common, well-known result, that a fine healthy infant is born at full time, which at the end of a month begins to snuffle, and a few weeks later shows a symmetrical rash closely similar to those seen in the acquired syphilis of adults. These symptoms are, as in the adult, amenable to treatment, and also subject to the law of spontaneous disappearance after a definite duration. It is remarkable that the period which intervenes between birth and the full development of the exanthem stage is exactly that occupied by incubation in the case of acquired disease, and the conjecture is forced upon us, that the development of the yeast in the child's blood is restrained during intra-uterine life, and commences only with the function of respiration. The phenomena which ensue are those of a specific fever, and in no respect resemble those of development. If aberrations from normal development are ultimately produced—peculiar physiognomy, malformed teeth, &c.—they are always in ratio with the preceding infantile inflammations, and are probably directly produced by them. If a syphilitic child in infancy escape syphilitic inflammation, its development will probably be in no respect peculiar; it may grow well, and present every appearance of excellent health. Thus far, then, we may assert, that what happens to a syphilitic infant is closely similar to what occurs in the acquired disease, and suggests rather breeding of specific poison in the blood and tissues, than the inheritance of "potentialities of development." It may be added that, were the latter creed the true one, we ought to find children inheriting the disease at the special stage to which it had attained in their parents; whereas, this old opinion is now generally discarded, and it is acknowledged that, whatever may be the stage in the parent, the child will begin again at the early secondary period. If the theory of yeast-contagion to the ovum be the true one, we ought to find no shading-off in degree of severity of transmitted taint—the child must inherit the whole of syphilis or none; whereas, if the other view be correct, we should expect the most various gradations, the severity being in relation with the parent's state. Now, I believe that the general—perhaps the universal—opinion held at the present time is, that inherited syphilis does really shade off, so to speak, in the younger children. On former occasions, I have myself used expressions favouring this

view, and, if I now discredit it, I fear I may encounter suspicion of allowing my theoretic belief to influence my interpretation of fact. I may, however, assure the Society, that, although I have admittedly changed my opinion on the suggestion of theory, I have not allowed myself to do so without collating very carefully the evidence afforded by the histories of some scores of syphilitic families. It appears to me, as the result of this study, that there is no proof of the transmission of minor degrees of syphilis, and that what we witness in children is rather exactly parallel with what we observe in the acquired disease, *very variable severity in different individuals.* If the real facts as regards the natural history of syphilis could be obtained, we should probably find that a considerable proportion of those who pass through the syphilitic fever after chancre-contagion, do so without rash, without sore throat, without, indeed, any visible symptoms, and that this is by no means always dependent upon the treatment, but rather upon some peculiarity in the patient's organization, or some arrest in the development of the yeast poison. If this be the case in the acquired disease, we need not wonder that many infants who yet show evidences of taint in later life escape the ordinary *rôle* of symptoms during the first few months of the first year. I have notes of many families in which some suffered and others appeared to escape; and although, as we should expect, it is the rule for the eldest to suffer most severely, there are so many exceptions to this, that I do not think we can infer anything as to diminished intensity of the poison. We ought rather to say, that it is the rule for the eldest to suffer most often, and that the chance of total escape is increased with each successive year. As the rule, the younger members of syphilitic families show no traces of the disease whatever, and appear to have escaped entirely. Lastly, it may be remarked, that if the inheritance of syphilis were an inheritance of "potentialities of development," and not, as Mr. Simon has well expressed it, of "a material something which passes absolutely and bodily into the infected ovum," we should expect to have mixed and ill-defined results. We should see the syphilitic potentiality mingling itself with others and producing hybrid diseases. And, further, one scarcely sees how on such an hypothesis a syphilitic parent should ever have healthy children, or why his younger ones should suffer less than his early ones. Admitting that the subject is one of much difficulty, and that further investigations of fact are needed, my conclusion still is for the present a tolerably confident

one, that the transmission of syphilis to children is like that from person to person in the acquired disease, a communication of special germs, and that there is no such thing as the transmission of less or more—that the fœtus either gets syphilis in the full or not at all.

I confess that it seems very difficult, taking this view of the mode of transmission, to believe it possible that the germs received *in utero* can survive long enough to be transferred to a third generation. For this to take place, a period of vitality of nearly twenty years must in most cases be presupposed, and this is, I believe, far longer than can be proved in the case of the acquired disease. Facts bearing on this point are very few in number, and most of them negative. Mr. Simon mentioned briefly the other night one which seemed to favour it. Some years ago I recorded a number in proof that those who had suffered severely might yet bear healthy children, and with them one which might be considered to bear in the other direction. The fallacies are, however, almost insurmountable.

Whilst urging that the kind of poison communicated in parental transmission is probably just the same as that conveyed in chancre-contagion, we must not forget that there are possibilities of difference, to fully appreciate which a knowledge of the laws of cryptogamic life, rather than of heredity, is needed. To this important topic Mr. Simon and Dr. Greenfield both alluded; but I shall be best able to illustrate what is meant if I may be allowed to pass back to the speech of Dr. Broadbent. Amongst certain difficulties in the way of accepting the theory that tertiary deposits occur only where secondary disease has preceded them, Dr. Broadbent mentioned the well-known opinion that a healthy woman pregnant by a syphilitic husband may acquire the disease from the fœtus, and pass at once to the tertiary stage, having never suffered from any secondary symptoms whatever. My reply to this would be, that in such cases the "tertiary symptoms" are never displayed early; that they follow, as in other instances, after long intervals; and that the cases are probably examples of the secondary stage being passed through without external signs. The subject is, however, far too important and too curious to be thus dismissed. If what seem to be facts respecting it be really credible, we appear to have a wholly novel phase of the disease opened out to us for investigation. It has long been believed by many that the mother might become diseased by absorption direct from the blood of the fœtus, and without the intervention of any chancre. It is just twenty years since I

wrote a paper on this subject, and collected a good deal of evidence concerning it. My conclusion was, as stated by Dr. Broadbent, that in these cases the woman never suffers from secondary symptoms, and experiences nothing during the pregnancy which infects her, excepting perhaps a little loss of strength and slight indefinite ailments. In further proof that she does really contract something, I made use of the well-known observation of Abraham Colles that syphilitic infants nursed at the breast often infect wet nurses, but never their own mothers. I did not, however, until within the last few weeks, fully appreciate the bearing of this fact. If Colles's law be one which has no exceptions, it follows that all women who bear syphilitic children contract syphilis; for how else can they obtain immunity? And, since it is notorious that women under such circumstances scarcely ever show secondary symptoms, it follows, further, that we have here a form of syphilis which is protective, but which is unattended by any cutaneous outbreak. Thus, syphilis acquired by blood-contagion from the fœtus would appear to be, for the mother, a parallel with vaccination in regard to smallpox: she gains immunity without suffering from any severe form of disease. The botanist will at once suggest that probably in both cases the explanation is to be found in heteromorphism or alternation of generations on the part of the fungus. To him the clinicist might aptly rejoin, that really one might almost have expected it; for, when the mother gets syphilis from the fœtus, she obtains it from fluids in which the plant-life is evidently under some very special restraint; for in the fœtus itself, as a rule, no development of it takes place during the nine months of intra-uterine life. We have only to suppose that the same condition of the yeast which existed in the fœtus is perpetuated in the mother, and the thing is done. We had no right to suppose that infection by inoculation of solids (or chancre-contagion) would be exactly the same in its results as direct imbibition by the blood.

As we may call vaccinia undeveloped yet protective variola, so we may consider syphilis, derived from the fœtus, as an undeveloped yet protective form of that disease; and we have here another most interesting point of analogy between syphilis and the exanthems.

Before, however, we accept as probable such a possibility as that just hinted at, it is desirable to look at the facts with the utmost incredulity. Let us doubt unsparingly at every stage of the reason-

ing. First, is Colles's law true? I cannot see any escape from the conclusion that it is. It was announced in 1837, and has received, I believe, the assent of every authority who has written on the subject since. It has attracted attention both at home and abroad, and I am not aware that a single exception to it has been recorded. We have all of us seen chancres on the nipples of wet nurses. They are, indeed, not very unfrequent. We have, however, none of us seen such on those of the mothers of infected children. Let us remember that it is very unusual to put a syphilitic infant out to wet nurse—a thing which no prudent surgeon would ever permit—and that probably, for one so nursed, a hundred are suckled by their mothers; and we shall appreciate the weight with which this entire absence of proof that mothers ever suffer bears. It amounts, I think, to all but proof that they are absolutely insusceptible. It is as strong in that direction as is the rarity of small-pox within short intervals after successful vaccination. We must remember also that these mothers of syphilitic infants not only nurse one infected child, but often several in succession; that they not only suckle them, but handle them, dress their sores, and in various ways through long periods expose themselves to risk. If it be granted that it is proved that these mothers—a very numerous class—have really in some way had syphilis and acquired immunity, I do not think there can be much dispute as to the next fact, that they do not during pregnancy show any of the usual symptoms of the disease in its secondary stage. This is a matter of everyday experience. There remains, however, the possibility that the syphilis may have been gone through prior to pregnancy; and I am well aware that the few remaining writers who teach that syphilis can be inherited only from the mother will hail this confirmation of Colles's law as a strong support for their opinions. Here, however, again I must appeal to everyday experience. Is it not the fact that women bear syphilitic children without having ever themselves, either before or during pregnancy, had any symptoms, either primary or secondary, of that disease? If this happened only once or twice, we might reasonably doubt the histories given us. But it is not so; it is in hundreds of cases; and few, I think, of much experience, can doubt that, as a rule, syphilis is inherited from the father; and that the mother never shows any external signs of the malady. I purpose shortly to publish the evidence which I have collected on this and some kindred subjects; and the assertion just made will, no doubt,

receive the scrutiny of other observers. If the argument should in the end be thought to be substantiated, some other very interesting questions will suggest themselves. We shall have to ask—What are the ulterior liabilities of a woman who has thus acquired the modified form of syphilis? Can the taint be transmitted from her to offspring borne subsequently to a healthy father? Is her blood at any period contagious; and what would be the result of inoculation with it? Does the taint in her last as long as when the disease is acquired in the ordinary way? I have already, in speaking of the difficulty of measuring the length of time during which it is possible for the tendency to transmit to offspring to last, adverted to the fallacy introduced by the fact that usually the mother acquires the disease from her first pregnancy, and may herself become the source of contamination in the second. We must not, however, take it for granted that a taint acquired in this peculiar manner is transmissible, or, at any rate, that it is so for any long period of time. It is quite possible that its stages may be far shorter than those of the common type of the disease.

Here, Mr. President, I will ask permission to leave the proper topics of reply, and to make a suggestion. It is this, that our next debate should be on the laws of evolution of the exanthemata. Surgeons have been asked to make a clean breast of it as regards syphilis; and we have told all that we know, and much, perhaps, that we only half know. Let us next solicit of medicine a similar declaration of faith in reference to the most interesting part of her proper domain. There are a host of questions upon which we surgeons, out of pure scientific curiosity, and with no wish to intrude into medical practice, are longing to be enlightened. By what right does diphtheria claim its place as a specific fever? If the rash of typhoid be not symmetrical, on what pattern and by what influences is it arranged? What is the interpretation of the curious phenomena of corymbiform variola? If a pregnant woman suffer from an exanthematic fever, what are the possible events as regards the fœtus? and does it ever happen under such circumstances that the infant developes the disease, as in the case of syphilis, soon after it gains access to the light? Are not the stages of the exanthemata far less accurately determined and much more liable to variation than is generally admitted? May not varicella, for instance, vary in its incubation period from fifteen to twenty-seven days? and in its exanthem period from four (the average) to fourteen or twenty days?

—limits of variation wider than we need claim for syphilis. Is it possible to convey the disease by inoculation with varicella-fluid?—a fact denied by most writers, but affirmed by one of the latest without citation of any evidence. Is it not probable that cases are very common in which patients go through specific fevers and acquire protection, without having had any rash or shown any external symptoms? On these and many other important topics medical opinion is, I believe, very far from being settled; and their investigation might, perhaps, have the collateral advantage of diverting the zeal of our medical friends (excepting in the *post-mortem* room, where they are always welcome) from the one specific fever, which is all that the surgeon can claim as his. To speak seriously, it has sometimes struck me forcibly that, in comparing syphilis with the exanthemata, the large amount of uncertainty which still surrounds the latter is a most serious bar to progress.

Amongst the more important of the questions which have been put to me in the course of the debate, I find one in the very suggestive speech of Dr. Greenfield, as to whether there may not be some after-results of syphilis which are to be classed rather as those of malnutrition than as consequences of the specific poison. He illustrates the question by asking whether all the children who suffer from keratitis have really had syphilis, and whether it may not, in some cases, rank rather as a defect in nutritive power. To this I may give a clear reply, that I believe that all the conditions which we have as yet recognised as syphilitic, whether in the acquired or the inherited form of the disease, are the direct results of the disease. A severe attack of syphilis may of course damage a man's nutritive power, but as the result of such damage we shall not encounter anything in the least special; and it is quite impossible that he should, in virtue of such, transmit to his children a state of cachexia rendering them liable to such a disease as interstitial keratitis. The latter malady cannot, I feel convinced, occur to any one who has not had syphilis. Interstitial keratitis also obeys the usual law of syphilitic inflammations in always showing tendency to spontaneous cure, which would scarcely be the case if it resulted from defect of nutrition. There is, however, a rare form of choroido-retinitis, which is steadily and slowly aggressive, much like a degenerative affair; but I suspect that it is secondary to damage done by previous inflammation, otherwise it might seem to be an example of what Dr. Greenfield is seeking.

The peculiar relationship in which the gumma, the soft sore, and phagedænic action, stand to syphilis, have perhaps been sufficiently discussed. I may note, however, that, with the exception, I think, of Mr. de Méric, no speaker has attacked the dogmatic assertion which I very purposely made as to my belief in the essential alliance between phagedænic and syphilitic inflammations. I was very anxious to attract attention to this subject, believing it to be of great clinical importance. I have long held, and publicly taught in the most positive manner, that, certain very rare cases excepted, all well-characterised phagedæna may be traced to syphilis; and that it is from the contagion by pus from syphilitic sores (mostly tertiary, and not containing the specific virus) that hospital phagedæna takes its rise. You, Mr. President, can, I know, give us some valuable evidence on this point; and as I hold that its knowledge is of great importance, in order to the prevention of epidemics of phagedæna, both in military and civil hospitals, I rather regret that it has not, on the present occasion, had the benefit of more criticism. Mr. de Méric had somewhat misunderstood my meaning, and seemed to think that I held syphilitic phagedæna to be incurable, which was very far from what I intended to say. I should be very glad if I could believe that the silence of other surgeons implied acceptance of my creed.

As a curious illustration of the difficulty in making one's meaning clear, I find that I have been criticised by Mr. Wood and others for attempting, as they think, to define too closely the stages of syphilis, whilst Mr. de Méric complains that I have shown too great a tendency to put the whole disease in one lump, and expresses his preference for the doctrine of stages. I cannot defend myself on this point from either critic without recapitulating almost the whole of my address—a task which the Society will, I am sure, gladly excuse me. Briefly, however, I may say that I consider the several so-called stages of syphilis as in part natural and in part conventional, and that I have not the slightest wish to make them more definite than is required by clinical convenience or suggested by pathological probability.

Amongst the chief contributions to facts, we must place Dr. Buzzard's valuable statistics as to the average age of those who suffer from disease of the nervous system, Dr. Hilton Fagge's statements as to the frequent alliance between syphilis and amyloid disease, and Dr. Greenfield's account of certain pathological details. Dr. Moxon

has also mentioned some interesting examples of symmetrical tertiary growths; and Dr. Greenfield has recorded a *post-mortem* examination in the secondary stage, in which gummata were found in the dura mater. I am sanguine that, hereafter, many of the topics which as yet have been only debated, will be examined by the light of accumulations of new facts, otherwise some regret might perhaps be felt that, following, perhaps, my own bad example, most speakers have contented themselves by expressions of opinion.

The possibility of diagnosing syphilis by the odour of the patient was adverted to by Sir William Gull. The late Mr. Wormald used to teach that this was possible, and was fond of saying, "They can't deceive me; I nose 'em." It probably requires a very good sense of smell to make much use of this symptom. For myself, I may confess that I have rarely been able to distinguish anything peculiar in the odour of adults suffering from syphilis; but in infants I think it is often very perceptible and quite special.

In answer to the numerous surgeons who have requested further details as to my disbelief in "dualism," I may briefly say that, although of course we must all fully recognise the differences between hard and soft sores, yet I do not think that the soft is a special form of disease. On the contrary, I believe that it is usually produced by contagion with the results of syphilitic inflammation; and that, if we could get rid of true syphilis, we should after a while cease to see examples of the soft sore also, with the exception of those which result from common inflammation, balanitis, and the like. My views on this point are explained in detail in a lecture published in the 'Lancet' six months ago. To those who were inclined to complain that I had said that dualism was "dead" whilst it is still mentioned in several standard and excellent works, I can only reply that I was careful to say only dead, and that I did not say that it was buried. Had I done so, I should have been a little premature.

The observation made in the address as to the extreme infrequency of affections of the brain and nerves in inherited syphilis has been confirmed by Dr. Buzzard and all speakers who have adverted to it. As none have mentioned facts in opposition, I think we may now regard it as generally admitted. I am not sure but that some statements which have been made may even help to explain it. Dr. Buzzard and Dr. Greenfield have both called attention to the probability that the tertiary growths take place in perivascular spaces and other adjuncts of the lymphatic system. Professor Sigmund of

Vienna has also, during this discussion, in a paper published in the 'Practitioner,' drawn attention to the circumstance that syphilis, from its beginning, shows an especial tendency to develope in lymphatic structures. Now, we must remember that this is true only of syphilis when acquired by a chancre. It is not the case when it is transmitted from parent to child, nor when a fœtus infects the blood of its mother. In these, there is in the beginning no implication of glands. This may possibly be the reason why in neither of these classes of cases do we find in the later stages of the disease that liability to peri-vascular growths and gummata which are not unfrequently witnessed as results of the chancre form of syphilis. It appears highly probable that we have to deal with syphilis in two or three (perhaps more) different moods, varying according to the precise manner in which the transference of the yeast-poison is effected, and the structures into which it is first introduced. In connection with these differing moods, different classes of sequelæ must be expected.

Dr. Wilks, Mr. Berkeley Hill, Dr. Buzzard, and others, have adverted to the interesting fact that serious tertiary symptoms are not unfrequently met with in those who have suffered but very little in the earlier stages. This is to be explained probably by several different considerations. In the first place, it is true only of a certain class. Some of the very worst examples of tertiary disease in which multiple manifestations occur, happen often to those who have suffered throughout very severely, who have perhaps scarcely ever got rid of their symptoms. Such at least has been my experience, and these are often patients with whom the specifics have disagreed, or who show, in particular, an idiosyncrasy as regards mercury. They are often very seriously out of health. There is, however, another class in which the tertiary symptoms attract much attention, and become very serious, not so much on account of their own extent or severity, but because located in an important position. A patient apparently in perfect health may have a single gumma on his brain, which may cause him most serious distress, whilst he has not about him another symptom of syphilis, either present or past. These are the cases to which I suspect allusion was made, and in these not unfrequently the patient knows but little about his early symptoms. I suspect that the real explanation of the frequency of such cases as these, and of the converse fact that those who have had well-marked secondaries usually escape sequelæ, is that the latter are well treated by mercury, whilst the former neglect it.

I am painfully aware that in this reply I have done very scant justice to the numerous speakers who have taken part in the debate. I have, however, I hope, profited by all, and have endeavoured to incorporate in my statements to-night the hints they have given. That I have not even alluded to the splendid speech of Sir James Paget, is to be explained by the fact, that we agree on almost every point; and this, again, finds its solution in the further fact, that he was my teacher, not alone, as I was proud to be reminded, in general surgery, but also in the special doctrines of syphilis.

In conclusion, Mr. President, I have to express my sense of obligation for the many valuable suggestions which have been addressed to me both in public and in private, during the course of this prolonged debate. I should be ungrateful, indeed, if I did not very warmly appreciate also the kind and very flattering manner in which my introductory remarks have been received as well by my friends in this room as by my critics in the medical press. It is not for me to say whether the debate will add greatly to our knowledge of its subject; but I may confess with pleasure that I have myself learned much. If it have not been a harvest, we may, I think, hope, at any rate, that it has been seed-time, and that syphilis, long ago named by one of our great masters "the key to all pathology," may in the future be found yet more useful.

REPORT OF THE COMMITTEE

OF THE

PATHOLOGICAL SOCIETY OF LONDON APPOINTED TO INQUIRE INTO THE MATTER OF DISPLACED, MOVEABLE AND FLOATING KIDNEYS.

The matter which has been referred to us for investigation is the displacement and unnatural mobility of the kidneys. It seems to us, however, that the state of knowledge of displacements of the kidneys is tolerably satisfactory, and that we can throw but little further light upon this part of our subject. Looking at the matter of kidneys unduly moveable, we think it well to divide them into two varieties. The first form is that in which the organ may be moved to some extent beneath the peritonæum. A certain degree of this mobility appears not to be very uncommon. It will be found to be present in a considerable number of subjects, if, as soon as the body be opened, the kidneys be handled without the removal of any of the organs. The amount of movement possible is not commonly greater than an inch upwards and downwards. Occasionally, however, a flaccidity of the peritonæum exists to a much greater extent, so as to allow the kidney to move under the peritonæum over a space described in one of the reports sent to us as a circle having a diameter of eight or nine inches. In the second variety, the peritonæum passes over the posterior surface of the kidney, forming a kind of meso-nephron.

From the evidence which has been laid before us it appears that the so-called floating kidney may depend upon either of the above states: that the peritonæum may be flaccid and loose to such an extent as to allow the kidney to move under it, so as to come in contact with the walls of the belly; or to leave its natural place, and pass to or below the brim of the pelvis; or, indeed, in some

cases to encroach upon the opposite side of the belly. A like moveableness or floating of the kidney may be due to the presence of a meso-nephron, already spoken of. It will, therefore, be seen that the terms moveable and floating kidney must not be used as strictly corresponding to two anatomical varieties, since a kidney without a meso-nephron may give rise to all the clinical phænomena shown by one with a meso-nephron. Both these anatomical varieties merge by insensible degrees into one another, and these two expressions, moveable and floating, can only be used as implying different degrees of one diseased state, which according to its extent may give rise to a slightly mobile or an extremely mobile kidney.

Cases of undue mobility of the kidney verified by examination after death have been several times recorded. One specimen was brought before our Society sixteen years ago by Mr. Durham.[1] Dr. Priestley has described a case, under the care of Sir James Simpson, in which after death the peritonæum was found reflected over the posterior surface of the right kidney, thus allowing great motion on the right side.[2] Other instances have been recorded by Mr. Adams,[3] Dr. Jago,[4] in which the state of the kidney was diagnosticated during life and verified by examination after death, Dr. Sawyer,[5] Girard,[6] Urag,[7] and others. The cases printed below contain abundant evidence of the facts stated above.

Those who wish still farther to pursue this subject will find the bibliography up to 1866 given in Rollett's 'Die Pathologie und Therapie der beweglichen Niere,' published at Erlangen in 1866.

Charles J. Hare.
J. S. Bristowe.
Samuel Wilks.
John Williams.

May 2nd, 1876. J. Wickham Legg, *Secretary.*

[1] Durham, 'Trans.' of this Society, 1860, vol. xi, p. 142.
[2] Priestley, 'Medical Times and Gazette,' March 14, 1857, p. 262.
[3] Adams, ibid., p. 651.
[4] Jago, ibid., 1872, vol. ii, pp. 328 and 409.
[5] Sawyer, 'Birmingham Medical Review,' 1872, April, p. 120.
[6] Girard, 'Journal hebd.,' 1836, t. iv, No. 53, p. 445.
[7] Urag, quoted by Fritz, 'Arch. gén. de Méd.,' 1859, vol. ii, p. 167.

Case of floating kidney. Post-mortem examination by Dr. Joseph Coates.

Mary M—, aged 54, single. The account in the ward journal is very meagre. She was admitted to Glasgow Royal Infirmary on November 21st, 1874, and died on November 25th. It is noted that a kind of tumour was felt at the margin of the right lobe of the liver, and she feels pain in it when pressed upon.

The following is extracted from *post-mortem* report:—"The right kidney is at once seen to be dislocated and moveable. The loose external capsule is slightly adherent to the lower surface of the liver, but, notwithstanding this, the kidney moves with wonderful freedom in the loose retro-peritoneal tissue. It can be pushed back nearly into its normal position, but the adhesion to the liver prevents it from altogether reaching it. Then it is easily moved forward quite up to the middle line, and its border can be made even to project beyond. In its movements the kidney keeps close to the lower border of the liver, so that as a tumour it might be taken to be related to the liver.

"Both kidneys are in the contracted stage of Bright's disease. They are reduced in size; the capsule is firmly adherent; the surface granular, and the cortical substance, as seen on section, reduced to a thin rind on the surface of the pyramids, not exceeding an average thickness of two lines.

"The liver is considerably flattened and depressed, its lower margin reaching more than an inch below the hypochondria. It presents the thickening of the capsule on the anterior surface, and the shallow groove running from left to right along this surface so common in females.

"The capsule of the spleen is thickened, but the organ is normal."

Case of moveable kidney (or kidneys, especially right). Mr. C. B. Keetley's case.

In the dissecting-room, St. Bartholomew's Hospital, February 19th, 1876.

Sex, female. Age, 68. Cause of death, unknown.

Condition of subject, fresh, and well injected with preservative; condition of nutrition, fairly nourished.

The right kidney lies at about its usual position, but can be moved freely in all directions. There cannot be said to be a mesentery, strictly speaking, for the two layers of peritonæum, forming what resembles a mesentery, are so independent of each other, and so far apart. But the peritonæum over the kidney moves with it; the kidney does not simply roll about in the fat behind the serous membrane.

Without straining any of the structures, or emptying any of the viscera, the lower end of the organ can be pushed down to Poupart's ligament, near the antero-superior iliac spine; while the upper end can be raised till it nearly overlaps (that is, more than touches) the anterior border of the liver and the edge of the costal cartilages. This latter movement can be done by placing the hand outside the abdominal wall in the loins, and pushing the kidney forwards; however, the skin and muscles are loosened by dissection. The outer border can be carried one and a half inches to the left of the middle line across the spine.

The left kidney is somewhat moveable, though not nearly so much as the right.

All the processes of the peritonæum, except the ligaments between the liver and diaphragm, are extremely long. Thus, the foramen of Winslow is very large (from the laxity of the gastro-hepatic omentum); and there are long ascending and descending mesocola. The great omentum is very large. There is no hernia.

Dr. Goodhart's cases.

Cancer of liver, and secondarily of lungs; occlusion of vena cava by pressure; moveable kidney (192, 66.) *Post-mortem examination* by Dr. Moxon.

July 2nd.—Dr. Gull. Mary.

Mary Johnson, 57, admitted June 4th.

The right kidney placed transversely, with its hilus upwards, and rather moveable, so that it would rise up half way over the spine on that side.

The liver was very large, 7 lbs., but bent upwards to chest rather than down into abdomen; still, it seems possible that it may have displaced kidney. No evidence one way or the other in the *post-mortem* report. There is no reference to any doubtfully renal tumour in clinical report (vol. ii, p. 375).

Epithelial cancer of pharynx; pneumonia; moveable kidney. (150, 39.) *Post-mortem examination* by Dr. Moxon.

June 1st.—Dr. Habershon. Lydia.

Anne E. Langabeer, 60, admitted May 12th.

On opening the abdomen the right kidney was found to be visible between the colon and the liver as a prominence of the size of an apple. It was very moveable and would pass from the natural position over to the left side of the spine behind the peritoneum beneath the mesocolon and into the root of or opposite root of the mesentery. Its figure was much altered, being small above, very bulbous below, and its position when at ease was just down to the level of the crista ilii by its lower margin.

P. 300, vol. xii, ref. to clin. report. No mention made of any abdominal trouble of any kind.

Old laryngeal disease; tracheotomy; chronic pneumonia; gangrene of lung; constitutional syphilis; moveable kidney. Post-mortem examination by Dr. Moxon.

May 7th.—Dr. Habershon. Esther.

Louisa Martin, 29, admitted April 17th. No clinical observation of the position of kidney.

Kidneys, 8 oz., very fatty. The *right* was singularly moveable. Its position drew attention on first opening the abdomen, for its lower end projected forwards, the organ having become horizontal in position. The colon allowed it to come into view by forming a renal flexure rather than an hepatic. The ascending colon was short and below the projecting kidney; the bowel bent to the left and came forwards, being then horizontal and having a mesentery three inches wide, rapidly widening. Such was the appearance, the kidney being at once in view projecting two inches below the liver and nearly on the same level, so that in replacing the anterior wall the tumour was easily felt. On examining the organ it was found with the peritoneum still *in situ*. The kidney would slip with the utmost ease to the left over the front of the spine so as to overhang the left lumbar region and downwards so as to reach the promontories of the sacrum or easily to lie in the upper half of the false pelvis, which, indeed, appeared to be a common position for it. Its lower end would reach

to half an inch from a line drawn across from the antero-superior spine of the ilium to the other. There was no mesenteric fold for it, but when lifted off the wall behind the peritonæum of course it raised a fold in front of it.

Certainly the kidney might, if it had drawn attention during life, have given rise to doubts as to the nature of the tumour which it must have formed.

Vol. viii, p. 78, reference to clinical report. Gives no note of any observation of abdominal tumour.

Elephantiasis of leg; amputation; exhaustion; displaced kidney, moveable. (417, 74.) *Post-mortem examination* by Dr. GOODHART.

November 13th.—Mr. Bryant. Lydia.

Jemima Curtis, 63, admitted November 4th, 1874.

On opening the abdomen the liver was large and several inches below the margins of the ribs. Pushing forwards below it and to the right of the gall-bladder was a prominent lump beneath the peritonæum, which proved to be the kidney. The chest was a very long one, and the liver squeezed down below it gave to the trunk an elongated appearance. Immediately below the liver was the kidney, with the ureter running across its present front surface downwards and inwards towards the median line. It was covered by peritonæum, as was also the posterior surface. It was thus turned completely over, and lying on the psoas and spine, with its anterior surface posteriorly. Its position now was an oblique one from above downwards and outwards. Its upper end behind the duodenum pushed the latter well forwards from the spine, so that the whole of that part of the bowel was exposed in front. The transverse colon lay below it, and the other coils were placed around and below the kidney, and between which its lower end peeped out and came to the surface. The kidney was not easily moved upwards or downwards, but very easily replaced in its natural position as regards its surfaces, anterior and posterior, *i. e.* it could be turned over and brought into a line vertical to long axis of trunk. The capsule lay $2\frac{1}{2}$ inches or more away from the kidney, in its natural position up under the liver. The kidney was of normal shape, the other side healthy.

Moveable kidney. Dr. PHILIP BINDLEY'S case.

This was a case of moveable right kidney. It would have been impossible to detect it during life, owing to the abdomen being distended with ascitic fluid.

The patient, a rather fat woman, aged 59, married, had been during infancy a victim to rickets, which had given rise to much deformity. The antero-posterior diameter of the lower part of the thorax was greatly increased, and the lateral regions had fallen in, giving rise to a deep groove on each side of the chest. The liver was not only depressed low into the abdomen, but was also greatly altered in shape, so that it rudely resembled a pear, with the big end to the right; and it was this globular right lobe, bulging down into the lumbar region, that had apparently pushed the kidney from its proper place. When the intestines were moved on one side the precise position of the kidney was easily made out. It lay between and partly covered by the transverse mesocolon and the mesentery, somewhat obliquely over the bodies and transverse processes of the third and fourth lumbar vertebræ, and slightly over the fifth, the hilus looking outwards. Its upper end overlapped the middle line by half an inch, while the lower end slanted off away from the middle line.

The peritonæum over it—there was no reflection over the opposite surface—was very loose, though not sufficiently so as to deserve the designation pouch; but a large fold of it could be pinched up between the fingers, in the same way that the skin on the back of the hand can, and it allowed the underlying organ to be completely grasped. The kidney felt quite slippery beneath the peritonæum, and slid about under the hand like the testicle slips about in the scrotum. A circle of eight or nine inches in diameter would represent the area over which the organ could be made to glide, and simple elevation of the buttocks or of one side of the body was sufficient to cause it to gravitate towards a lower point.

When a slit was made into the peritonæum, which was thickened by a layer of subperitoneal fat, and the cut edges raised, a loose areolar tissue was found connecting it with the renal capsule, long enough and loose enough to allow of the kidney being moved in every direction without returning. No recess existed in the right loin to indicate the place from which the kidney had been dislocated;

on the contrary, fat had accumulated there, and had shaped itself to the under surface of the depressed liver. The kidney had carried no fat away with it, nor did the supra-renal capsule appear to have been disturbed, with the exception, perhaps, of being a little lower and a little nearer the middle line than usual. The kidney had been rolled over on itself, so that its convex edge looked towards and lay on the bodies of the vertebræ, and its posterior surface became anterior, the vessels hooking round to enter at the hilus. The peritonæum over the fat in the right loin did not hang loose, but was intimately connected with it, and this rendered it impossible to roll the kidney back into its normal position. The right renal vessels were half an inch longer than the left. The ureter was twisted on itself and lengthened and dilated. In the pelvis of the kidney was a large uric acid calculus, which weighed exactly 100 grains. Both kidneys were "granular contracted."

Moveable kidney. Dr. Tuckwell's case.

A woman, aged 45, was for years an inmate of the Warneford Asylum, near Oxford, suffering from chronic mania. Cause of death, acute phthisis. On opening the abdomen the right kidney was plainly seen lying immediately below the liver, with its lower end in front of the spine, extending a line or two beyond the mesial line of the abdomen; its concavity turned upwards towards the pyloric end of the stomach, which lay in close contact with it; its upper end turned to the right and concealed beneath the right lobe of the liver. It could be moved backwards, pushed upwards and to the right, but could not be moved more to the left than the position it already occupied. Directly the liver was removed the right kidney fell back by its natural weight into its natural bed; it had therefore been clearly displaced, and kept out of its place, by the right lobe of the liver. The chest was long and narrow, the lower ribs compressed by former tight lacing. The liver was similarly indented pretty deeply, along the usual line, by tight lacing. The kidney, then, seemed to have been pushed forwards, and at the same time to have its lower end twisted round to the left. The supra-renal capsule had moved with the kidney, and occupied its usual relation to the kidney.

On feeling through the abdominal walls, before the organs were displaced, the right kidney could be felt as a distinct tumour of the

shape and size of the kidney, but not so freely moveable as in other cases of supposed floating kidney which I have felt during life. It was, however, like enough to other cases to make me feel pretty sure that I have felt during life, though not been so fortunate as to see after death, other cases exactly similar. I should add that the left kidney occupied its natural position, and that both kidneys were healthy.

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